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ESOPHAGEAL HIATUS DIAPHRAGMATIC HERNIA

ETIOLOGY, DIAGNOSIS, AND TREATMENT IN 123 CASES*

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FELLOW members of the American Thoracic Association. Your selection of me as president of our society is a great honor which I fully appreciate and for which I am most grateful. Any address that I could deliver to you would be inadequate, in my estimation, fully to meet the requirements of this high office. I have selected as my subject, nevertheless, the etiology, diagnosis, and treatment of esophageal hiatus diaphragmatic hernia, a subject in which I have been greatly interested for the past ten to twelve years. The subject is of increasing general interest and although much advancement has been made in the last few years in knowledge of the subject, many fundamental problems are still unsolved. I shall avail myself of the privilege of my office and invite your free discussion of the presentation of this subject.

Herniations of abdominal viscera through the esophageal hiatus are the most common types of diaphragmatic hernia occurring in adult life. These hernias are of considerable general interest because of the relative frequency of their occurrence, their indefinite etiology, the progressive character of their development, the varied and complex symptoms produced by them and because of their treatment, which may be conservative if the hernias are small and the symptoms mild, but which may embrace surgical intervention if the hernias are large.

Many different schemes of terminology and classification, based on etiology, pathologic anatomy, roentgenologic findings or clinical findings, have been suggested for hernias through the esophageal hiatus.

*Presidential Address read before the Twenty-First Annual Meeting of the American Association for Thoracic Surgery, at Atlanta, Ga., April 4, 5, and 6, 1938.

The multiplicity of terms which have been used undoubtedly has led to considerable misunderstanding as to the various types of hernia found. I believe that in classifying these hernias an actual description of the findings should be made so that there will be no misunderstanding as to the structures involved. I believe that all esophageal hiatus hernias are essentially congenital in origin in that there is a congenital malformation of the esophageal hiatus and of its attachments to the lower end of the esophagus and to the cardiac portion of the stomach. It is probably rare that these hiatus hernias are present at birth, although I have operated on four children, less than 11 years of age, who had large hiatus hernias. The symptoms of two of these children dated from the first days of nursing from the breast, which would indicate that the hernias had been present at birth. Hiatus hernias usually are not recognized and do not present symptoms until later in life. In my experience they have been most commonly noted in the decade of life between 50 and 59 years; I have operated on forty-six patients who were in this decade of life. However, patients may not present symptoms until in the seventh or eighth decade of life.

I believe it is best to classify all true hernias through the esophageal hiatus under the one term "esophageal hiatus diaphragmatic hernia." True esophageal hiatus hernias can be divided into two types. In the first type of esophageal hiatus hernia the esophagus is of normal length and its lower end is not elevated above the diaphragm, but a portion of the stomach has herniated into the posterior mediastinum, beside the esophagus. This type of hernia is often designated as a para-esophageal hiatus hernia. The hernias are usually small to moderate in size and rarely involve more than one-fourth to one-half of the cardiac end of stomach, which is contained in the hernial sac. They constitution about one-third of the esophageal hiatus hernias on which I have operated (Figs. 1, 2, 3, and 4).

The second type of hiatus hernias is composed of those in which the esophagus is of normal length but its lower end is elevated above the level of the diaphragm, and the herniated stomach is in the posterior mediastinum. These hernias are usually of larger size than those of the first type and a third of the stomach or the entire stomach, with a portion of the omentum and occasionally a portion of the colon, is within the hernial sac. These hernias usually fill the entire mediastinum and generally project into the left thoracic cavity but they may project into the right thoracic cavity or into both thoracic cavities. These constitute about two-thirds of the esophageal hiatus hernias on which I have operated.

I believe that the essential difference between these two types is one of degree rather than any fundamental difference in origin. Inasmuch as these hernias are progressive in character and are in reality a sliding type of hernia, hernias of the first type may ultimately develop

into those of the second type as more of the stomach becomes included in the hernial sac. In many cases of esophageal hiatus hernias there is a dilatation of the lower end of the esophagus. This is usually more marked in the larger types of hiatus hernias.

Fig. 1. Fig. 2.

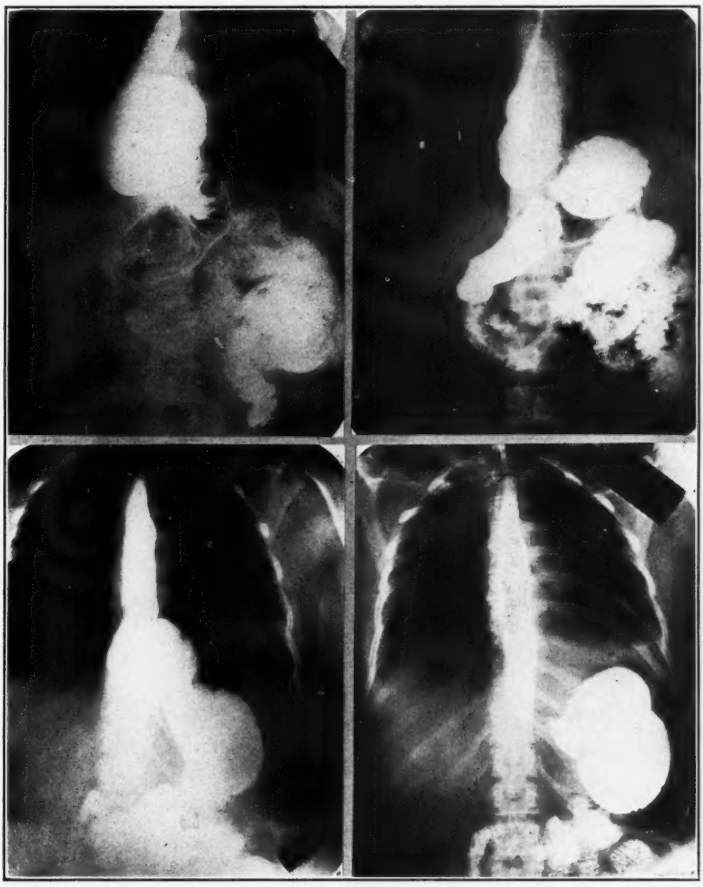

Fig. 3. Fig. 4.

Fig. 1.—Herniation of two-thirds of cardiac end of stomach into posterior mediastinum and extending into right thoracic cavity. Marked displacement of esophagus to right.

Fig. 2.—Same case as that represented in Fig. 1, three weeks after operation. Entire stomach in normal position below elevated left portion of diaphragm. Esophagus normal.

Fig. 3.—Herniation of about a fourth of cardiac end of stomach through esophageal hiatus into posterior mediastinum.

Fig. 4.—Same case as that represented in Fig. 3, two weeks after operation, showing stomach in normal position below diaphragm. Left portion of diaphragm is slightly elevated from preliminary temporary interruption of left phrenic nerve.

With the more frequent roentgenologic recognition of hiatus hernias in the last few years, a third group has been designated (Figs. 5 and 6). In this group, there is a small projection of the cardiac end of the stomach through the esophageal hiatus, considered by some writers to be attributable to an incompetent esophageal hiatus. This condition is more commonly seen among elderly than among younger people. I do not believe that these should be considered true hernias. Their distinction from true hernias and their definite recognition usually can be made by means of esophagoscopic examination in conjunction with roentgenologic examination. Abnormalities of this third group may be considered potential hiatus hernias. They probably are attributable to atrophy of elastic fibers of the diaphragmatico-esophageal

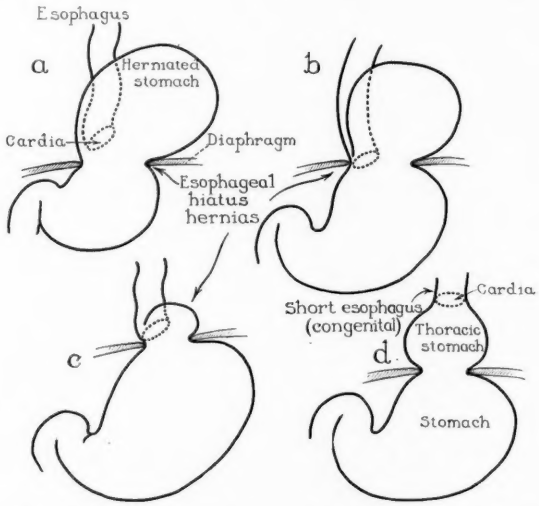

Fig. 5.—*a*, Esophageal hiatus hernia in which there is herniation of greater portion of the stomach through an enlarged esophageal hiatus into the posterior mediastinum and associated elevation of the esophagus. *b*, Esophageal hiatus hernia, with herniation of portion of cardiac end of stomach through enlarged esophageal hiatus. Esophagus in normal position. Para-esophageal hernia. *c*, Slight protrusion of cardia and lower part of esophagus above the diaphragm; so-called incompetent hiatus. Not a true hernia. *d*, Congenital shortening of esophagus, with partial thoracic stomach. Not a true hernia.

membrane, together with relaxation of the esophageal muscle ring; such atrophy and relaxation commonly occur in elderly people. I have never operated in a case of this type and I do not believe that this condition requires surgical treatment. However, elderly people may have definite esophageal hiatus hernias that may produce sufficient symptoms to demand surgical intervention. I have repaired hiatus hernias of twenty-three patients who were more than 60 years of age. Three of these patients were more than 70 years of age. One of these three presented extensive gastric erosion along the lesser curvature at the cardia, which shows that these hernias, when they

afflict elderly people, are essentially the same as those found among younger individuals in that they are of a sliding type; the same complications may develop as those often found among patients who are younger.

In another type of hernia, because of congenital malformation, the esophagus is not long enough to permit the stomach to reach its normal position below the diaphragm. The stomach is held suspended in the posterior mediastinum above the diaphragm. In this type of case the abnormality is often considered to be an esophageal hiatus hernia, but I do not believe it is a true hernia. I believe the condition could be better described as a "congenitally short esophagus with partial

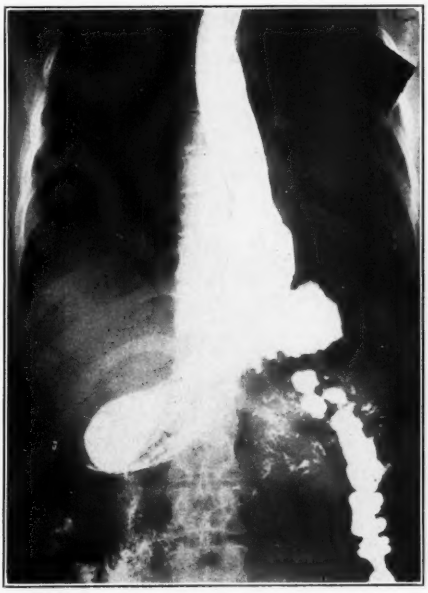

Fig. 6.—Not a true hernia but a relaxation of the esophageal hiatus with slight protrusion of cardiac end of stomach and moderate dilatation of lower end of esophagus.

thoracic stomach." In my experience this condition rarely has been found at operation. I have operated on five patients who presented all the roentgenologic signs of short esophagus but at the time of operation I found that in four cases the esophagus, although its lower end was above the diaphragm, was of sufficient length to reach below the diaphragm when the herniated stomach was reduced and the diaphragm elevated by interruption of the left phrenic nerve. In one case it was impossible to bring the stomach entirely below the elevated diaphragm and it was necessary to suture a small portion of the cardia into the hiatus when the hiatus was closed. This indicates that it is not always possible by roentgenologic methods to determine the

type of hernia present. I believe that this condition, although rare, is an entity but should not be grouped with the true hiatus hernias (Figs. 7 and 8).

The condition of short esophagus, from an etiological standpoint, falls into two groups: first, those cases in which the esophagus is congenitally short, as described above, and second, those cases in which ulceration has caused cicatricial contraction of the lower end of the esophagus. The cicatricial contraction draws the cardia of the stomach above the diaphragm, and the condition usually is associated with stenosis of the lower part of the esophagus.

In considering cases of short esophagus from a surgical standpoint, it is important not to confuse them with true hiatus hernias, for their surgical management is entirely different. In cases of true hernia, the

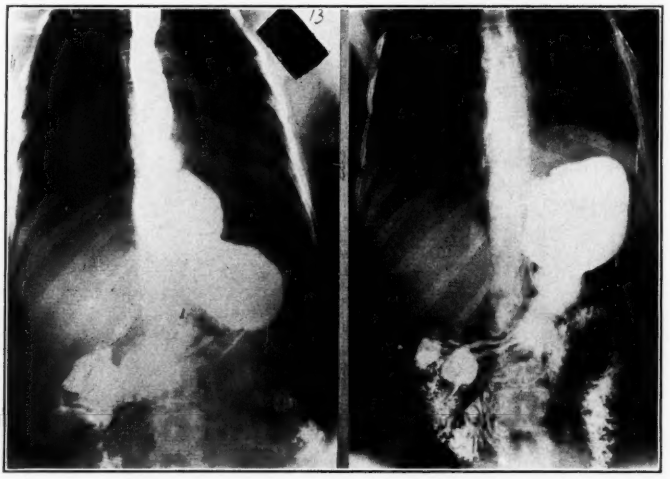

Fig. 7. Fig. 8.

Fig. 7.—Congenital short esophagus with one-fifth of cardiac end of stomach above diaphragm.

Fig. 8.—Same case as that represented in Fig. 7. One month after repair of hernia. Entire stomach in normal position below diaphragm.

surgical problem is that of replacement of the herniated viscera, removal of the hernial sac, and repair of the abnormally enlarged esophageal hiatus. In cases of congenital short esophagus the surgical problem is that of reconstruction and elevation of the diaphragm to a position above the stomach; this can be accomplished if the congenital shortening of the esophagus is not too great. In those cases in which the stomach is elevated above the diaphragm because of traction of cicatrices following ulceration, the symptoms usually are relieved by dilatation of the stenosed esophagus.

Many writers believe that hiatus hernias are of congenital origin, although some attribute them to certain types of trauma and to senile

changes in the tissues about the hiatus. However, because of the relatively few elderly people who present hiatus hernias roentgenologically, compared to the number of patients whose stomachs are examined roentgenologically, and considering the number of elderly patients of whom I have examined the esophageal hiatus at the time of other operations and have not found a hernia present, I believe that the fundamental cause of the hernia, whether the patients are elderly or young, is to be found in the embryologic formation of the diaphragm. The congenitally defective hiatus is not able to withstand the abnormal, and in some instances the normal, pulsion and traction effects of intra-abdominal and intrathoracic pressure. The stress on the hiatus does not have to be great in order to produce hernia. Less than 10 per cent of the patients who were proved to have hiatus hernias at operation gave histories of injury. In 50 per cent of these cases some symptoms antedated the injury; this suggests that some degree of herniation had been present prior to the injury.

ANATOMY

Before considering some of the embryologic factors in the formation of the diaphragm, I shall review briefly the normal relationship between the esophageal hiatus and the lower end of the esophagus and cardia. The esophageal hiatus is normally situated to left of the medial line, at the level of the upper border of the tenth thoracic vertebra, in the dorsal part of the dome of the diaphragm. The muscular elements that form the muscle ring of the hiatus usually originate from the median portion of the right crus, but the hiatus may be formed by the crossing of both right and left crura after they have united in front of the aorta to form the aortic orifice. This muscle ring of the hiatus loosely approximates the lower end of the esophagus but is not directly attached to it. Fixation of the diaphragm to the lower end of the esophagus and to the cardia of the stomach is obtained by the diaphragmatico-esophageal membrane, which consists of elastic and fibrous tissue originating in the fascial coverings of the diaphragm, mostly those of the abdominal side but partly those of the thoracic side of the diaphragm. This elastic and fibrous membrane has its origin lateral to the hiatus muscle ring over which it passes, with slight attachments, to fill the space between the inner margin of the muscle and the esophagus; it is inserted into the lower 2 or 3 cm. of the esophagus and into the upper 1.5 to 2 cm. of the stomach, in a fanlike manner. This elastic and fibrous membrane constitutes a flexible connection between the diaphragm, esophagus, and cardia, permitting a certain amount of mobility during the act of swallowing and during respiratory movements. Its greatest strength is in its esophageal attachment, where it acts as an antagonist to the longitudinal muscle fibers of the esophagus, preventing these muscles from pulling the cardia of the stomach through the hiatus (Fig. 9).

The lower part of the esophagus passes obliquely through the diaphragm and extends below the hiatus for a varying distance of from 0.5 to 1.5 cm., where it joins the stomach at an angle. This junction is the true cardia and is always normally below the diaphragm.

The normal peritoneal covering and attachments of the esophagus and cardia of the stomach, around the esophageal hiatus, are of great importance. The abdominal portion of the esophagus is loosely covered with peritoneum on its anterior and left aspects. The uppermost part of the stomach is loosely covered, for about 2 cm. anteriorly and 3 cm. posteriorly, where the peritoneum fuses with the serous covering of the gastric wall. The union of these two folds of peritoneum forms the gastrophrenic ligament which fixes the cardiac end of the stomach to the diaphragm and continues to the left to form the phrenicolienal ligament. Beneath this loose peritoneal covering there is a triangular

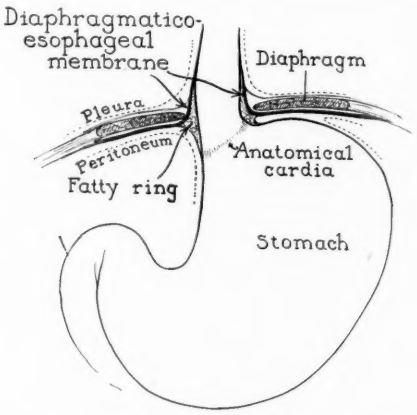

Fig. 9.—Normal anatomic relationship between lower end of esophagus, cardiac end of stomach and esophageal hiatus, showing the relationship of the diaphragmatico-esophageal membrane to these structures.

pad of fat and connective tissue and the attachments of the diaphragmatico-esophageal membrane. This loose peritoneal attachment around the esophageal hiatus permits flexible movement of the esophagus without pulling on the peritoneum.

The diaphragmatico-esophageal membrane and the loose attachment of the peritoneum at the cardia have considerable bearing on hiatus hernias. It is probable that the hiatus hernias of elderly people, which are accompanied by insufficiency of the hiatus, are attributable to atrophy of this protective elastic membrane, particularly in cases in which there is an abnormally large hiatus. The elastic membrane has been sufficient during the prime of life to protect the opening but, with the atrophy associated with advancing years, the stomach is permitted to protrude through the large hiatus to a position above the diaphragm. It is probable in these cases that this would have oc-

curred earlier in life had abnormal stress been applied, such as trauma, respiratory difficulty, or marked increase in intra-abdominal pressure. The elastic and fibrous membrane forms one of the coverings of the hernial sac in the smaller types of hernia. In a number of cases I have removed specimens of the tissue outside of the peritoneal lining of the sac and have demonstrated the elastic tissue of this membrane by microscopic examination after special staining of the tissues. In large hernias this membrane seems to be pushed forward and there is no barrier to the peritoneal sac which enters the posterior mediastinum. In these cases the entire stomach often herniates into the thorax (Fig. 10).

The loose attachment of the peritoneum around the hiatus may be a factor in the formation of hiatus hernias as it permits the beginning of the herniation of the cardiac end of the stomach and formation of the hernial sac.

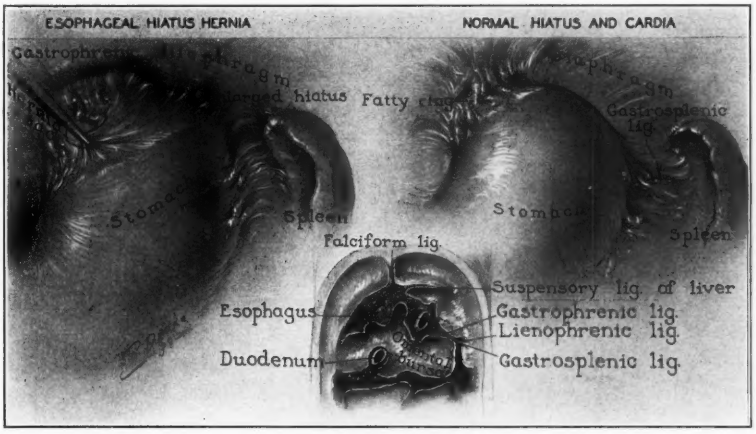

Fig. 10.—Relationship of peritoneal coverings of cardia, fatty ring, peritoneal ligament, stomach, and spleen, when stomach is in normal position and when there is an esophageal hiatus hernia. Insert: Normal relationship of omental bursa and peritoneal attachments to posterior abdominal wall and diaphragm.

In all true hernias this loose peritoneal fold fuses with the serosa farther down on the cardia of the stomach than normal. The peritoneum does not extend around the greater curvature of the stomach to the posterior wall to form the gastrophrenic ligament but fuses with the serosa of the anterior wall, so that there is no gastrophrenic ligament and a very short phrenicolienal ligament. This places the spleen close to the left margin of the hiatus, thus permitting the upper part of the cardiac end of the stomach to be abnormally free posteriorly, beneath the enlarged esophageal hiatus.

The consistency with which the surgeon finds abnormal attachments of the peritoneal fold and of the gastrophrenic ligament to the cardiac end of the stomach, and abnormal enlargement of the muscular ring

of the esophageal hiatus, suggests that this type of hernia results from delay in descent of the esophagus as related to the formation of the lumbar portion of the diaphragm (Figs. 11 and 12).

In practically all of the 102 cases of hiatus hernia in which I have effected repair, the esophageal hiatus has been enlarged. In many cases this enlargement of the hiatus is not in direct proportion to the

Fig. 11.

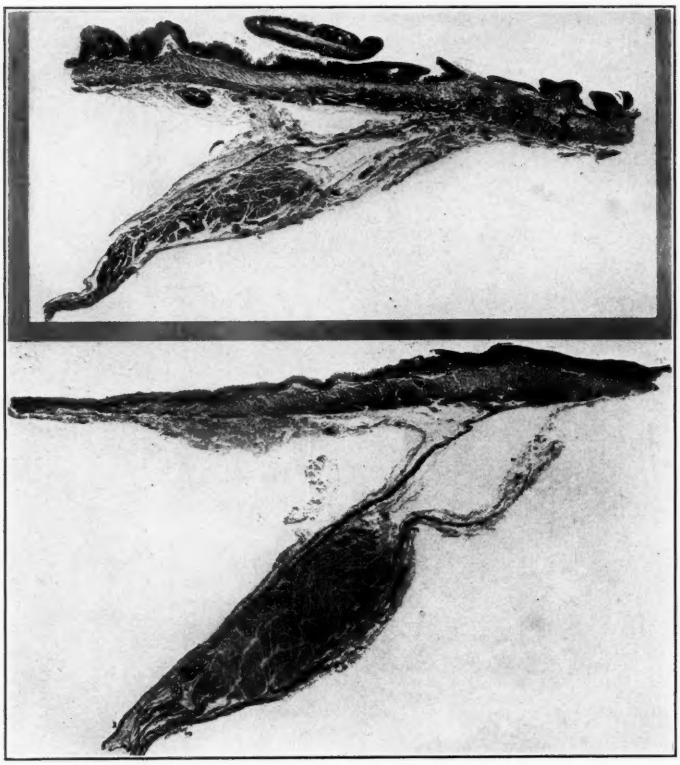

Fig. 12.

Fig. 11.—Especially stained specimen, showing normal attachments of the diaphragmatico-esophageal membrane to esophagus and stomach.

Fig. 12.—Especially stained specimen, showing abnormal attachments of the diaphragmatico-esophageal membrane of an esophageal hiatus hernia of moderate size. There is a wide space between muscle and esophagus, with marked thinning of the diaphragmatico-esophageal membrane after it has become somewhat contracted in fixation.

amount of stomach which has herniated. In some cases I have found the entire stomach to have passed through a relatively slightly enlarged hiatus. In these cases the hernial opening is usually in the posterior part of the muscle ring, and in some cases the hernia extends into the right thoracic cavity. The peritoneal sac seems to originate

posteriorly from the omental bursa, suggesting that a persistent infracardiac bursa, communicating with the right dorsopleural recess, may have been the cause of the hernia.

EMBRYOLOGY

The formation of the diaphragm from embryologic structures is a highly complex process, as the component parts of the diaphragm are derived from several sources. That the anterolateral (sternocostal) parts of the diaphragm are formed from fusion of the septum transversum and pleuroperitoneal membrane is universally accepted, but there is considerable difference of opinion among embryologists as to the derivation of the tissue elements and as to time of completion of the posteromedian (lumbar) portion of the diaphragm. Some embryologists are of the opinion that this lumbar portion of the diaphragm is derived from mesodermic cells of the receding wolffian body and the dorsal mesentery.

The transverse septum which forms the anterolateral portion of the diaphragm of the adult arises from mesoderm in the upper part of the cervical region. Its descent is gradual in the early weeks of embryonic life and at the 5 to 6 mm. stage the structure is opposite the fourth and fifth cervical segments, where it derives its innervation from branches of the third, fourth, and fifth cervical nerve roots.

In 7 mm. embryos the septum, with the pleuroperitoneal membrane, begins to migrate caudally, rapidly passing the pulmonary buds and the anlage of the stomach. This places the greater portion of the lung in the pleural cavity and the anlage of the stomach partially above the diaphragm.

In the 9.4 mm. embryo, the liver has become firmly anchored to the right dorsal wall of the body of the plica vena cava. The midgut begins to grow more rapidly and the yolk-sac disappears. This frees the intestine from the umbilicus, and rotation of the bowel begins.

In the 11 mm. embryo, the stomach, which has been practically stationary, now follows rapidly behind the descending septum transversum and at 17 mm. it has virtually reached its permanent position. This descent is made possible by the sudden elongation of the esophagus. During this descent the right and left dorsopleural recessi are converted into bursae, which surround the cardia. The left bursa usually disappears, and the right, when well developed, is known as the infracardiac bursa and bears a definite relation to hiatus hernia.

About 11 mm., a physiologic alteration in the abdominal region takes place. The celom has not increased in size enough to accommodate the rapid changes that are taking place within its walls and which are augmented by the descent of the septum. As Mall stated, "Since the liver grows downward and crowds upon the rapidly elongating intestine, the intestine must escape it if it has a chance and the celomic space within the umbilical cord naturally receives it." This

phenomenon, known as physiologic umbilical herniation, provides a reservoir for the abdominal content until the peritoneal cavity has grown sufficiently to store its own content. The herniated intestine is restored to the abdominal cavity by the growth of the abdominal wall past the fixed mesentery of the intestines. This physiologic herniation starts at 11 mm., is well developed at 22 mm., and starts on its way toward voluntary reduction at about 35 mm.

During this period of greater activity in the abdominal celom, the diaphragm is incompletely formed and the hiatus pleuroperitonealis is patent. Closure takes place on the right side first at about 17 or 18 mm. and on the left side at about 19 to 20 mm. The left side of the liver has largely disappeared, and the whole organ has rotated to the right and fused with the right wall of the body, protecting to a great extent the right half of the diaphragm and probably aiding its more rapid completion. The stomach has reached its permanent site below the diaphragm before the left half of the diaphragm is closed. Therefore the hiatus pleuroperitonealis, patent on the left side, is in direct contact with the rapidly forming and constantly changing hollow viscera during this period of physiologic umbilical herniation.

It would seem that this time of incomplete closure of the diaphragm, in relation to multiple rapid changes which are taking place in the embryo, is of great importance in the preponderance of left-sided congenital defects. To recapitulate, these changes consist of: (1) the occurrence of normal physiologic umbilical herniation, (2) the patency and closure of the hiatuses connecting the peritoneal and pleural cavities, the left closing last, (3) the sudden descent of the diaphragm considerably in advance of descent of the stomach, (4) the shift of the liver to the right because of anchorage and the degeneration of the left lobe from vascular change and pressure, (5) the presence of hollow, mobile viscera on the left side, (6) the assuming of an elevated position and smaller size of the left lung, (7) the rotation of the stomach to the left, and (8) the presence of bursae at the esophageal opening.

In the foregoing it is seen that embryonic herniation would occur most commonly in the embryo at from 11 to 20 mm. Congenital herniation of somewhat later formation can be explained on many of the same grounds. It is owing to failure of parts of the diaphragm to mature or to excessive degeneration of muscle elements in the formation of the central tendon.

Inasmuch as the stomach descends behind the septum transversum, if there is a delay in descent of the stomach, the lumbar portion of the diaphragm will be imperfectly developed, and the esophageal hiatus will be formed around the cardiac end of the stomach instead of around the esophagus. This will result in an abnormally large

hiatus with deficiency both in the muscle ring and in the attachments of the diaphragmatico-esophageal membrane. The degree of the deficiency depends on the amount of gastric anlage in the thorax at the time of muscularization of the lumbar portion of the diaphragm. In rare instances the stomach may remain in the elevated position as a result of a congenitally short esophagus and a partial thoracic stomach will result. In most instances the esophagus continues to elongate normally, thus placing the stomach below the diaphragm. This abnormal position of the stomach will cause an abnormal enlargement of the esophageal hiatus to be produced, with a wide space between the margin of the muscle and the wall of the esophagus. It will also result in imperfect fixation of the elastic membrane to the esophagus and stomach and an abnormally large peritoneal fold extending well down on the cardia of the stomach. This abnormal relationship will permit much more flexibility of the esophagus in the enlarged hiatus than is normal. The more defective the formation of the hiatus, the more likely is herniation of the stomach to occur through it in later life. The true congenital hernia present at birth may be explained in this same way, but it is probable that persistence of the dorsopleural recess may be a factor in the origin of congenital hernias.

INCIDENCE OF HIATUS HERNIA

The incidence of esophageal hiatus diaphragmatic hernia is probably no greater now than it was twenty years ago. At that time the condition was thought to be uncommon and its recognition in life was rare. More frequent recognition of the condition in the last two decades has been owing primarily to development of roentgenologic methods of diagnosis. This more frequent recognition is exemplified by reviewing cases encountered at the Mayo Clinic from 1908 to 1938. During this period esophageal hiatus hernia was diagnosed roentgenologically or at operation in 416 cases. The first case in which hiatus hernia was found at operation was encountered in 1908. The condition was first recognized roentgenologically in 1921; in this case existence of the condition was not proved by operation. From 1908 to 1926 the diagnosis was made in seventeen cases, in five of which the patients were treated surgically and in twelve they were not treated surgically. From 1926 to 1938, the diagnosis was made in 399 cases, in 123 of which treatment was surgical and in 276 of which surgical treatment was not employed. This shows that the condition was recognized in more than twenty-three times as many cases in the last twelve years as in the previous eighteen years. The marked increase in the number of cases in which the diagnosis was made in recent years indicates that recognition has increased in frequency with the advancement made in roentgenologic methods of diagnosis. The frequency of roentgenologic recognition is increasing year by year, as is shown by reviewing the cases in which the diagnosis was made

roentgenologically in 1937. In this year alone hiatus hernia was diagnosed in ninety-two cases while in 1921 the condition was recognized roentgenologically in only one case.

The relative incidence of the occurrence of esophageal hiatus hernia to other types of diaphragmatic hernia, in my experience, is shown by a review of 161 cases of diaphragmatic hernia in which I operated from 1926 to 1938. In this series there were 123 cases of esophageal hiatus hernia, four cases of congenital pleuroperitoneal hiatus hernia, two cases in which the hernia was owing to congenital absence of a portion of the left hemidiaphragm, two cases of hernia through the foramen of Morgagni, and thirty cases of traumatic hernia through the dome of the diaphragm. This shows that hernias through the esophageal hiatus, treated surgically, were between three and four times as common as all other types of hernias combined (Table I).

TABLE I

DATA CONCERNING SITUATION, ETIOLOGY, AND CONTENT OF HERNIA

SITE OF OPENING, TYPE	CASES	ETIOLOGY	CASES	CONTENT OF HERNIA	CASES
Esophageal hiatus	113	Congenital: (history of trauma, 12)	113	Stomach (omentum)	108
				Stomach and colon	5
Short esophagus	10	Congenital	10	Stomach only	10
Hiatus pleuroperitonealis	4	Congenital	4	Colon and small bowel	3
				Colon, small bowel, stomach, and spleen	1
Absence posterior fourth of left hemidiaphragm	2	Congenital	2	Stomach, colon, small bowel, and spleen	2
Foramen Morgagni (substernal)	2	Congenital	2	Colon and omentum	2
				Stomach only	5
Left hemidiaphragm	29	Trauma: (indirect injury, 20) (direct injury, 5) (inflammatory necrosis, 4)	29	Stomach and colon	9
				Stomach, colon, and small bowel	4
				Stomach, colon, and spleen	2
				Stomach, colon, spleen, and small bowel	6
				Stomach, colon, small bowel, and liver	2
				Stomach, colon, small bowel, spleen, and liver	1
Right hemidiaphragm	1	Trauma: (indirect injury)	1	Stomach, duodenum, colon, small bowel, liver (gall bladder), and head of pancreas	1
Totals	161		161		161

This study does not take into account the 276 cases in which esophageal hiatus hernia was recognized roentgenologically during the same period but surgical treatment was not employed. In comparing

the number of cases of hiatus hernia in which treatment was surgical (123 cases) with the number of cases in which treatment was conservative (276 cases), it would seem that only a third of the hernias recognized roentgenologically are considered to warrant surgical treatment. This is not entirely true, for in this series of 276 cases forty-two patients presented definite symptoms but surgical treatment was either contraindicated, deferred, or refused by the patient; in eighty-four cases the hernia was small or of moderate size, and symptoms were not sufficiently severe to warrant operation at the time of examination; therefore, the patients were placed under observation and may possibly require operation later. In 114 cases roentgenologic examination indicated that the hernias were small and symptoms were indefinite; thirty-six patients presented no clinical symptoms of hernia, and operation was not considered. In these two latter groups the roentgenologic finding of hernia, in many instances, was made incidentally, in the course of general examination, and there was no reason to consider surgical treatment.

SYMPTOMS

The symptoms of esophageal hiatus hernia depend on the amount and type of abdominal viscera herniated, the size and character of the enlarged esophageal hiatus, as well as the degree of interference with normal function of the diaphragm and intrathoracic organs. The subjective manifestations vary from complete lack of symptoms to very severe symptoms causing complete disability. In many instances small hernias, which involve only a portion of the cardiac end of the stomach, may produce more severe symptoms than larger hernias which involve a much larger portion of the stomach.

The symptoms vary a great deal from complete absence of symptoms to complete disability, and although they do not uniformly depend on the size of the hernia they often progress in severity and character with the increase in size of the hernia. The most important clinical consideration with reference to esophageal hiatus hernia is the frequency with which the symptoms simulate other organic disease of the abdomen and thorax; in many instances several clinical diagnoses can be made in the same case because of the changing symptomatology. Accordingly, the condition may be termed the "masquerader of the upper abdomen." In a study of this series of 123 cases it was found that an average of three previous erroneous clinical diagnoses had been made before the correct diagnosis was established. The most common erroneous diagnoses, in order of frequency, were cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperacidity, secondary anemia, cardiac disease, carcinoma of the cardia, stricture of the esophagus, appendicitis, and intestinal obstruction. In twenty-one of these cases the patients had been operated on previously for other conditions, without complete re-

lief of symptoms, and were completely relieved following repair of the hernia. Of these twenty-one patients, thirteen had undergone cholecystectomy previously; five, cholecystostomy; two, pyloroplasty; and one, gastroenterostomy.

The chief symptoms of hiatus hernia are pain, distress, gaseous eructation, vomiting, dyspnea, hemorrhage, weakness, anemia, and palpitation of the heart. The symptoms may begin at birth or at any time in life. The symptoms of these hernias are more uniform than those of other types of diaphragmatic hernia and often produce a fairly definite clinical syndrome over a clinical course of many years. No one case will include the entire clinical picture, but study of the clinical course in a large series of these cases has suggested the composite clinical syndrome now to be described.

The clinical syndrome is that of intermittent and usually progressive incarceration and obstruction of the stomach. At the onset the attacks are usually mild; they consist of epigastric distress that is projected through to the back and which comes on in the course of, or shortly after, a heavy meal; but they may be brought on by taking anything into an empty stomach, such as a cupful of coffee. The attacks are usually similar in character but vary a great deal in intensity, depending on the amount of incarceration and fixation of the stomach in the hernial opening. They are usually relieved by belching of gas and vomiting and are commonly considered to be attributable to cholecystitis, for which the patient is usually treated. As more of the stomach becomes incorporated in the hernia, the attacks become more severe; the pain is projected straight through to the back and to the lower left side of the thorax, is more marked to the left of the spinal column than elsewhere in the back, and often appears between the shoulder blades. This pain may be agonizing and difficulty is experienced in belching of gas and vomiting because of spasm of the diaphragm and reflex cardiospasm. The spasm of the diaphragm produces an hourglass deformity of the stomach, which interferes with emptying of the upper loculus and causes increased intragastric pressure. The pressure of the herniated portion of the stomach on the lower part of the esophagus interferes with belching of gas or vomiting. Spasm of the diaphragm is commonly associated with phrenic pain which is referred to the left shoulder and which at times may extend down the left arm. The increased pressure within the thorax causes cardiac embarrassment, with palpitation and tachycardia. Pressure on the lung and interference with motion of the diaphragm cause dyspnea. These symptoms are augmented when the patient lies down, and in the more severe cases it is necessary for patients to sit up to breathe. The attacks may last for from a few minutes to several hours and not uncommonly are considered to be caused by myocardial disease, relative to which the most frequent diagnosis is that of angina pectoris. The attacks often recur immediately after food has been

taken. There is often an interval of weeks or months between attacks. It is probable that during the interval between attacks the stomach is not incorporated in the hernial ring and is in its normal position below the diaphragm. When the attacks become more or less constant, the stomach usually has become fixed in the thorax by adhesions. All the early symptoms of pressure are augmented during attacks. There is loss of weight from inability to retain food and from marked restriction in diet because of the fear of bringing on an acute attack of gastric obstruction. This may be termed "food-fear."

The vomiting is more severe during attacks and often is of the retention type. During the severe vomiting the vomitus may contain blood. If the attacks are of long standing, the patient not uncommonly has a burning sensation in the epigastrium after meals, which is relieved by taking small quantities of food. If a large amount of food is taken it may bring on one of the attacks that is associated with incarceration of the stomach. Many of these patients present a fairly good syndrome of ulcer, are given medical care, and obtain partial relief because they have taken a restricted amount of food at frequent intervals.

Hemorrhage is not a common symptom; it usually is indicative of severe incarceration, with fixation of the stomach in the thorax. The bleeding is caused by erosion or ulceration of the mucous membrane, resulting from the forceful pressure exerted during the attacks of vomiting on the large, distorted, and congested stomach and to the trauma caused by the hernial ring as the stomach is forced in and out of the opening. This erosion may be superficial or, in cases of long standing, definite ulceration may form from repeated trauma. This is usually the final stage of incarceration. I have never seen a stomach that was strangulated as a result of hernia nor do I believe that strangulation is possible because of the powerful musculature and rich blood supply of the gastric wall. Because of the fixation of the stomach in the thorax, the obstruction may be complete and the vomiting may be so severe as to produce gastric tetany.

Patients who have esophageal hiatus hernia, of which the predominating symptoms are those of esophageal obstruction, are of particular interest and require careful clinical study. The symptoms may be attributable to an entirely unassociated lesion of the lower part of the esophagus, such as cardiospasm, carcinoma, or diverticulum, or they may be the result of ulceration or stricture of the esophagus caused by the hernia. This esophageal ulceration produced by the hernia is attributable to the repeated or constant pressure of the hernial sac on the lower part of the esophagus or to the regurgitation of food or gastric secretion into the lower part of the esophagus. The ulceration may be a small, localized lesion or it may involve the entire circumference of the lower part of the esophagus and later may contract and

produce stricture. Because of the possibility of there being a lesion in the lower end of the esophagus, caused by or unassociated with the hernia, I believe esophagoscopic examination is advisable in all cases.

SURGICAL TREATMENT

The treatment of esophageal hiatus hernia depends to some extent on the size and type of the hernia as well as on the severity or mildness of subjective symptoms and on the complications if any are present. In a general way these cases of hernia may be divided into three groups: (1) those cases in which there are no symptoms and in which, usually, the hernias are small; in these cases no treatment is indicated; (2) those cases in which there are moderate symptoms and the hernias are of moderate size; in many of these cases conservative treatment such as regulation of diet and reduction of weight is sufficient to relieve the symptoms; (3) those cases in which there is no response to conservative measures; in these cases usually the hernias are large and in many, in my experience, there are complications, such as incarceration of the stomach or gastric erosion. In these last cases the only treatment that assures relief of symptoms is operative repair of the hernia. In all cases in which a third or more of the stomach is involved in the hernia, surgical intervention should be considered because the condition is progressive and usually becomes rapidly worse after the hernia has attained this size. Operation should be performed before severe incarceration, with consequent obstruction and traumatic lesions of the stomach, has occurred. The operative risk is increased by gastric retention, and the technical difficulties are enhanced by fixation of the stomach to the diaphragm and to the hernial sac within the thorax. All cases in which the colon is involved in the hernia demand early operation because of the danger of intestinal obstruction.

Interruption of Phrenic Nerve.—Paralysis of the diaphragm, produced by temporary or permanent interruption of the phrenic nerve, is of value as a procedure preliminary to radical operative repair of many types of esophageal hiatus hernias. It is a necessary procedure in the surgical treatment of partial thoracic stomach resulting from a congenitally short esophagus. In some cases in which radical operative repair is contraindicated, it may be used as a palliative measure.

In most instances in which interruption of the phrenic nerve is utilized as a procedure preliminary to radical operative repair of the hernia, I prefer, first, to perform temporary interruption of the nerve by crushing it, because in many instances it may not be necessary for the paralysis to be permanent. Function is usually re-established in from three to six months. In cases in which re-establishment of function of the diaphragm is not desirable because of the danger of recurrence of the hernia, the paralysis can be made permanent by cutting or

evulsing the phrenic nerve. As a procedure preliminary to radical surgical treatment, interruption of the phrenic nerve is often of value in treatment of incarcerated and strangulated hernias because it prevents spasm of muscle and causes relaxation of the hernial ring.

Permanent paralysis of the diaphragm is rarely necessary in radical surgical treatment of esophageal hiatus hernias. In the 102 cases in which I have repaired the hernia, the phrenic nerve was permanently interrupted in only two cases and temporary interruption was performed in sixty-two.

Interruption of the phrenic nerve may be utilized as a palliative measure in esophageal hiatus diaphragmatic hernias when radical operative repair of the hernia is contraindicated because of the patient's condition. The purpose of this procedure is to prevent spasm of the diaphragm, which is the cause of the severe attacks of incarceration of the stomach in the hernial sac. I have performed this procedure in seven cases in which radical operation was contraindicated because of the general condition of the patient. Five of these patients had myocarditis and cardiac compensation was poor, and three of the five were more than 70 years of age. The sixth patient came to the clinic primarily because of renal colic; it was necessary to remove one kidney and function of the remaining kidney was not good. The symptoms of the seventh patient, an infant, were markedly relieved following phrenicotomy, and the parents did not wish the child to be submitted to radical operation.

Phrenicotomy, as a sole procedure, does not completely relieve the symptoms. There is always a moderate amount of gastric distress immediately or shortly after meals, but the patients get along fairly well if they are careful with their diet. This procedure is not applicable to hernias in which a large portion of the stomach is in the thorax, causing marked pressure on the heart and lungs, nor is it applicable in any case in which the intestines are involved in the hernia. The procedure should not be employed when radical repair can be effected. I have performed temporary interruption of the left phrenic nerve in fourteen cases, as a therapeutic test, to see if the symptoms would be relieved by this more simple procedure before considering radical repair of the hernia.

I wish to emphasize that permanent interruption of the phrenic nerve is rarely necessary and should never be done in cases of hiatus hernia until it is definitely ascertained that it is not advisable to reestablish the function of the diaphragm and that this procedure cannot replace operative repair of the hernia. This always should be done unless there is some contraindication to radical operative repair.

Radical Surgical Repair.—I prefer cyclopropane as the anesthetic agent, administered by means of a machine, under positive pressure, with a closed mask. In some instances it is necessary to utilize the intratracheal method of administration.

I prefer the abdominal approach in all cases of esophageal hiatus hernia because the herniated viscera are contained in a sac in the posterior mediastinum and do not enter the true pleural cavity. An oblique incision is made in the left rectus muscle and peritoneum, extending to the ensiform cartilage. The technical difficulties of adequate exposure of the esophageal hiatus are often considerable because of fixation of the left lobe of the liver to the leaf of the diaphragm. The exposure of the hiatus is greatly facilitated by cutting the suspensory ligament and retracting the left lobe of the liver to the right. This can be accomplished, when the left lobe is small, by folding it on itself and, when it is large, by retracting it forward into the wound. The spleen is often very adherent to the posterior part of the diaphragm and hernial openings but usually can be separated from these structures by blunt dissection. It is retracted posteriorly by a specially constructed retractor. In some instances the spleen may be almost drawn into the hiatus and may be so traumatized by separating it from its peritoneal attachments that it is advisable to remove it. This occurred in two cases of this series, in one of which tuberculosis was present in the spleen. All hernias through the esophageal hiatus have a hernial sac. The attachment of the sac to the stomach must be separated and the sac either completely removed or permitted to retract into the posterior mediastinum. I believe that this is one of the most important technical considerations in the surgical treatment of these hernias.

After the sac has been removed, the enlarged esophageal hiatus is repaired by overlapping the margins of the opening. Closure is usually made to the left of the esophagus, but in some cases it is necessary to close, partially, both to the right and left of the esophagus. In a few instances the enlargement of the esophageal opening is posterior, extending to the spinal column and requiring overlapping of the margins posterior to the esophagus. In such cases, the condition is often thought to be a herniation through the aortic opening, but usually extending over the aorta there is an imperfectly developed fibrous band which is the margin of the defective esophageal hiatus. The closure is usually made with living sutures of fascia lata which are removed from the thigh. The overlapped margins of the hernial opening are first stabilized with interrupted linen sutures. The fascia lata is then woven into the tissues by continuous suture and fixed in the tissues with interrupted linen sutures.

Before closure of the defective esophageal hiatus is completed around the lower part of the esophagus, it is important that a stomach tube of large caliber be placed through the esophagus into the stomach to aid in reconstruction of the normal esophageal opening and to prevent constriction of the esophagus by a tight closure. A small portion of the esophageal wall is incorporated in the innermost margin of the closure by a suture of chromic catgut.

Not uncommonly in these cases there is associated traumatic erosion or regional gastritis in the herniated portion of the stomach, along the lesser curvature, close to the cardia, and this erosion is often adherent to the margins of the hernial opening. Great care should be used in replacing the stomach in the abdomen and in removing the sac from the stomach because of the danger of perforating this thinned-out portion. In cases in which the ulcerated portion is penetrated, it should be repaired immediately with continuous catgut and linen sutures. Perforation occurred in two cases of this series, and the perforated portion was immediately repaired and the patients had an uneventful convalescence.

The abdomen always should be thoroughly explored for any other lesion, particularly of the stomach or gall bladder. In some cases it may be necessary to operate on other associated lesions, as occurred in four of my cases. In one of these cases it was necessary to perform gastric resection because of a perforating ulcer high on the lesser curvature, which was probably secondary to a traumatic erosion; in the second case the patient had a high gastric ulcer at the cardia, impinging on the lower part of the esophagus, and I performed posterior gastroenterostomy as well as repair of the hernia; in the third and fourth cases posterior gastroenterostomy was done for obstructing duodenal ulcers. I do not believe it advisable to carry out any additional surgical procedure at the time of repairing the hernia unless it is imperative, but it is well to know if the patient has gallstones or any other lesion in the upper part of the abdomen which might account for subsequent symptoms.

POSTOPERATIVE MANAGEMENT

All patients are placed in an oxygen cabinet or in an oxygen chamber immediately after operation. Most patients are given fluids intravenously for the first two or three days. All patients who present dilatation of the stomach at the time of operation are given intermittent or continuous gastric lavage to keep the stomach empty the first three to five days after operation. In many cases there is considerable shock during or immediately following the operation. If the blood pressure falls to less than 80 mm., the patient should be given a transfusion of blood or a solution of acacia should be injected intravenously. This is also advisable in all cases in which there is associated secondary anemia. The blood of every patient is grouped for transfusion before operation. In some cases there is traumatic effusion in the pleural cavity. Treatment of this effusion depends on the amount of respiratory embarrassment associated with it. In most instances the effusion is slight; it gradually will become absorbed and special treatment is not required. If the effusion progresses to produce respiratory embarrassment, pleurocentesis, one or more times, may be required. In my experience empyema never has occurred in patients in whom the

hernia was repaired by the abdominal approach. In some cases atelectasis may develop because of accumulation of mucus in the bronchi. In most cases this condition will respond to conservative treatment but in some it may cause serious symptoms and bronchoscopic aspiration may be necessary.

REVIEW OF THE OPERATIVE PROCEDURES EMPLOYED AND THE RESULTS OF OPERATION IN THE 123 CASES IN WHICH TREATMENT WAS SURGICAL

The operative procedures in the 123 cases on which this paper is mainly based were as follows: In 102 cases the patients were treated by radical operation; the herniated abdominal viscera were replaced in the abdomen, the hernial sac was removed, and the enlarged esophageal hiatus was repaired. In sixty-four of these 102 cases the diaphragm was either temporarily or permanently paralyzed preliminary to operative repair of the hernia. In sixty-two of the sixty-four cases the paralysis was temporary and was produced by crushing the nerve; in two cases of the sixty-four the paralysis was permanent and was effected by cutting the nerve. In the entire 102 cases the abdominal approach was employed.

In six cases it was necessary to perform other operative procedures at the time of repair of the hernia. In one of the six cases gastric resection of the Polya type was done for a gastric ulcer on the lesser curvature of the stomach. In three cases of the six, posterior gastroenterostomy was performed, in one case for high gastric ulcer involving the lower end of the esophagus, and in two cases for large duodenal ulcers causing almost complete obstruction of the pyloric end of the stomach. In two cases of the six, splenectomy was performed. In both of these the spleen was firmly adherent to the margins of the hernial opening and partially incorporated in the sac. The trauma to the spleen associated with its removal from the hernial orifice necessitated its excision. One of the removed spleens was tuberculous.

In five cases moderate shortening of the esophagus was associated with the hernia. In four of these five cases the diaphragm could be sutured entirely above the stomach after the diaphragmatic muscle had been paralyzed by phrenicotomy. In the fifth case a small portion of the cardia was incorporated in the closure of the hernial sac.

In twenty-one cases the left phrenic nerve was interrupted as a palliative or therapeutic measure; in seven of these cases it was the only procedure contemplated, for radical operation was contraindicated, and in the remaining fourteen the procedure was carried out as a therapeutic test. It may be necessary later to perform radical repair of the hernia in these patients to obtain complete relief of symptoms.

There were six deaths following operation. Five deaths were attributable to pulmonary congestion, or to pneumonia with respiratory

and cardiac failure. One of these five patients had cardiac disease and one cardiorenal disease prior to operation. The sixth death was attributable to cerebral embolism.

Recently I have reviewed the results obtained in the 117 cases in which the patients recovered from operation. Of the 117 patients, twenty-one were treated by interruption of the phrenic nerve and ninety-six by radical repair of the hernia. Of the twenty-one patients treated by interruption of the phrenic nerve, eleven were markedly relieved of symptoms, five were partially relieved of symptoms, and five obtained no benefit from the procedure. The two groups of five patients each had had indefinite symptoms or fairly large hernias and radical operation was contraindicated at the time of examination. Some of these patients will require repair of the hernia before relief of symptoms is obtained. Of the ninety-six cases in which the patients recovered from radical repair of the hernia, in four a small recurrence has developed. These recurrences occurred from three months to five years after operation. In two of the four cases the recurrence followed an influenzal type of pneumonia, which was accompanied by severe attacks of coughing. In the other two cases of the four no definite cause for the recurrence could be ascertained. These patients had gained considerable weight, which probably was a factor in the recurrence. In three cases the symptoms were mild and have been relieved somewhat by conservative treatment. In the fourth case the symptoms have remained moderately severe, and surgical repair of the recurrent hernia will be necessary to relieve them. In ninety-two cases in which the hernia was repaired by operation there has been no recurrence of the hernia, and the patients have been completely relieved of their symptoms. All patients have been examined roentgenologically every six months to one year after operation.

As was noted in this paper, twenty-one of these patients had undergone previous operations for the same complaint, without relief of symptoms, but were completely relieved following repair of the hernia.

TECHNIQUE, INDICATIONS, AND MAINTENANCE OF EXTRAPLEURAL PNEUMOTHORAX*

M. Olivier Monod†
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EXTRAPLEURAL pneumothorax is the most advanced form of surgical apicolysis.

Surgical therapy of pulmonary tuberculosis has passed through a number of successive stages.‡ Patients who come to the surgeon are gravely ill. At first it was necessary to demonstrate that they could be cured by thoracoplasty. It was then shown that if the indications were strictly conformed to, the operation could be performed without danger or fear of complications. Later, in lesions of the apex, the more conservative thoracoplasty was substituted for the total thoracoplasty. This was a distinct advance, and the extrapleural pneumothorax was also conceived to render a more conservative operation. It is the least mutilating and the most satisfactory operation that we possess to effect the surgical collapse of tuberculous lesions.

In this new operation the freeing of the upper lobe of the lung, apicolysis, is complete, smooth, and uniform, quite comparable to the collapse of intrapleural pneumothorax. (Attention is called to the similarity in the terminology of these two operations.§)

The monograph which we now publish is the fruit of our personal experience. It gives our personally elaborated technique. Our experience has led us to modify a number of points of the operative technique and to fix certain stages. We have observed some hitherto unknown variations in the course of the disease, and the method of maintaining the pneumothorax described below is the one which we have been able to elaborate slowly. Our conception concerning the indications differs greatly from that of foreign authors. We therefore request that the imperfections and the length of this study be excused. It is based upon the study of fifty patients and is the first complete work which appears on the subject.

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*Work of the Clinic of Professeur agrégé G. Lardennois Laënnec Hospital.

†Surgeon of the Marmottan Foundation and formerly chef de clinique of the Paris Medical School.

‡The name of my teacher, Dr. A. Maurer, is so intimately associated with the successive stages of the development of the doctrine of surgical treatment of pulmonary tuberculosis that I desire to place under his patronage this article, which reflects in so many respects his daily teaching.

The work of Robert Monod, Bonniot, Ameuille, Soupault, Fruchaud, Dreyfus-Le Foyer, Iselin, to mention only French authors, has added important contributions to these problems.

§We shall designate artificial extrapleural pneumothorax by the letters P.A.E. and the artificial intrapleural pneumothorax by the letters P.A.I.

PRINCIPLE OF THE METHOD

The primary aim of all surgical collapse therapy directed against upper lobe lesions is to free the apex of the lung. Apicolysis has been performed in a number of ways: scaleniotomy, resection of the first rib, thoracoplasty, etc. Thoracoplasty, as performed particularly by Dr. Maurer, with or without extrapleural cleavage, has given excellent collapse of the apex.

The advantages of P. A. E. over other surgical procedures are that it frees the lung more completely and with less traumatism. It differs from pneumolysis (apicolysis with plombage) in that a greater area of lung tissue can be collapsed, and once it is established it can be maintained more easily.

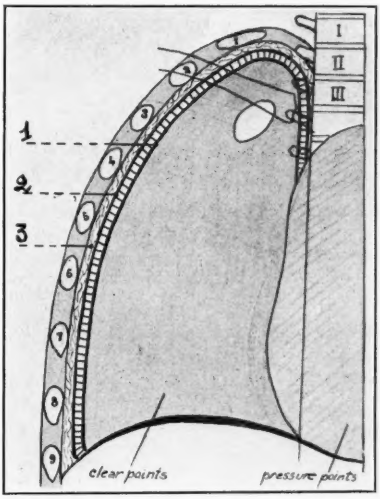

Fig. 1.—Diagram of an apical cavity with adhesive pleurae. *1*, Visceral pleura; *2*, parietal pleura adherent to *1*; *3*, subpleural space (or extrapleural space).

The P. A. E. is used only in adhesive lesions. Naturally, if the pleural cavity is completely free, intrapleural pneumothorax is the operation of choice. But if the pleurae are adherent, or if only the apex remains adherent, the P. A. E. enables one to free the lung and obtain a result quite analogous to that obtained with the P. A. I. To effect this, the parietal pleura is peeled away surgically from the bony thorax; access is thus had to the line of cleavage that normally exists between the parietal pleura on the one hand and the periosteum of the ribs and the perimysium of the intercostal muscles on the other hand. This plane of cleavage is located in the subpleural cellular tissue (endothoracic fascia). Thus the apex of the lung remains covered by the two adherent pleurae; the hand, under guidance of the eye, frees the apex from all the organs to which it is fixed: ribs, intercostal spaces, vertebrae, and mediastinal

viscera. When the operative wound is closed the pocket thus formed must be maintained by refills at regular intervals just as in the case of the usual intrapleural pneumothorax.

However, surgical cleavage is not always possible. A peripleuritis may be an obstacle, and it is difficult to know beforehand whether an important peripleuritis exists or not. It has been said that the older the lesions the greater the peripleural sclerosis. This is not always true. Certain young cavities may already be nondetachable, whereas it is frequently possible to detach pulmonary apices after years of disease. It has seemed to us that apicolysis that was difficult or impossible to perform has been due to lesions which showed a recent exacerbation, al-

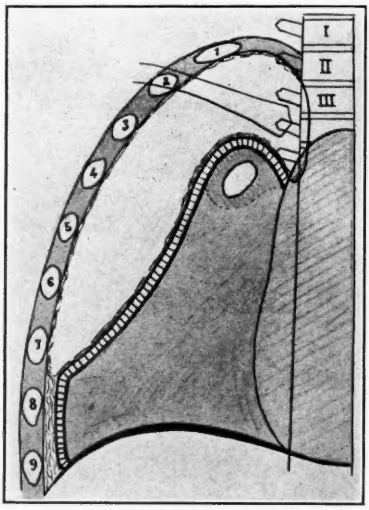

Fig. 2.—Artificial extrapleural pneumothorax immediately after operation. The detachment extends far down to the seventh or eighth rib laterally. The retracted apex of the lung has maintained its general form. The cavity is clearly seen, in the center of a more dense zone.

though the lesions themselves were of long standing. Does not general pathology teach us that it is impossible, for example, to extirpate an acute bartholinitis or a highly infected sebaceous cyst, whereas three weeks later, in the chronic stage, these lesions may be removed much more easily by cleaving the surrounding tissue?

TECHNIQUE

The description that follows represents only our personal technique. It is possible to do an extrapleural pneumothorax by the posterior approach (resection of the fourth rib) or by the anterior approach (resection of the second rib). A rib resection of 8 to 10 cm. in length allows enough room for all the necessary surgical maneuvers.

The posterior approach is the one most frequently employed because the great majority of lesions are situated centrally and posteriorly. Also, the closing is easier and is attended with less sequelae. However, if the cavity is situated near the anterior costal arch, it is advantageous to use the anterior approach. The detachment should be begun where the chances for the failure of collapse are greatest, for in the event of an anterior lesion, for example, it is useless to have first cleaved posteriorly, laterally, and superiorly, only to find at the end of the operation that the detachment of the anterior zone is impossible or dangerous and that the lung cannot be freed in the most important area, i.e., opposite the lesion.

Anesthesia.—The patient is operated on in the sitting position. He is held in this position by the apparatus that Dr. Garcia and the author have devised.

A local anesthesia (novocain at 0.005) is always sufficient. It has decided advantages: it conserves the cough reflex and expectoration, allows better control

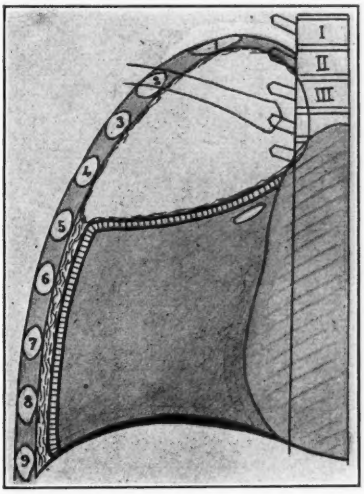

Fig. 3.—Extrapleural pneumothorax a few weeks after operation. The inferior part of the detached area has become reattached, whereas the floor of the pocket, i.e., the apex of the lung is depressed.

of the physiologic reactions of the patient (especially during the mediastinal detachment), obliges the surgeon to operate with all due care and caution.

To obtain a perfectly painless cleavage it is necessary to secure perfect anesthesia of all the neighboring tissues. It is thus advisable from time to time to infiltrate the muscles and block the spaces which surround the rib.

Incision.—A straight line incision is made running from the spinous process of D^2 (4 cm. below the prominence) to a point 2 to 3 cm. below the spine on the vertebral border of the scapula. This incision leads directly to the fourth rib. If the shoulder is drawn well forward the scapula will uncover a sufficient portion of the fourth rib. This incision is most physiologic. The trapezius is separated from its attachments medially and cut laterally. The rhomboideus is separated without being cut and the superior posterior serratus is separated from its attachments. During the first stage of the operation it is advisable to traumatize the muscles as little as possible, thus later facilitating the closing of the wound.

Rib Resection.—The fourth rib is carefully rugined so as to obtain even periosteal flaps that can be used when suturing the wound. The internal section of the rib should be on a line with the transverse process. At least 8 cm., preferably 10 to 12 cm., of the rib are resected. The deep aspect of the periosteum is then incised. It is separated from the parietal pleura by only a thin layer of subpleural tissue (endothoracic fascia). The incision in the periosteum should not be as long as the rib section and should stop short of it about 1 cm. This precaution prepares the way for an easier and tighter closing of the muscular wall.

Décollement.—With the periosteum incised one then proceeds to the décollement. If this proves easy, the finger and later the electrically lighted blunt-edged detacher is easily introduced between the costal vault and the parietal pleura which will cave in of itself. However, one should proceed slowly and cautiously, allowing the lung to collapse spontaneously. A rapid apicolysis may provoke serious shock.

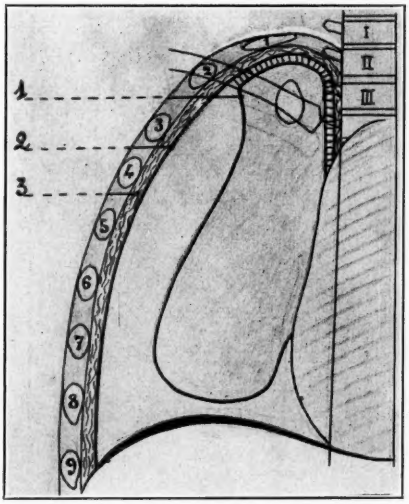

Fig. 4.—Diagram of a poor pneumothorax with apex adherent. *1*, visceral pleura; *2*, parietal pleura; *3*, endothoracic fascia (subpleural cellular tissue, extrapleural space).

The detachment is thus performed on all sides. On the mediastinal side, where the adhesions are more frequently localized, the plane of cleavage is not easy to find, and the detachment presents greater difficulties: the normal anatomic adhesions of the pleura to the head of the ribs and to the vertebrae offer no obstacle. Pathologic adhesions due to a peripleuritis are, however, more resistant. These may be localized, or arranged in bundles, or be fan-shaped, and give the impression of intrapleural adhesions seen by pleuroscopy. The peripleuritis, however, may be generalized, and to so great an extent that it becomes impossible or dangerous to continue the cleavage. One should especially guard against brutal maneuvers or the use of sharp-edged instruments to force a passage, for the dangers of hemorrhage, perforation, suppuration, etc., are always present.

Closing the Wound.—To close the periosteal wound a catgut stitch is employed. The following procedure can be of use in enlarging the ends of the wound: A periosteal incision is made on the outer surface of the ribs situated above and below the wound. The superior periosteal flap is scraped in a downward direction

and the inferior flap in an upward direction, thus permitting a greater play of the intercostal muscles that limit the wound.

The separated rhomboideus is closed by a stitch which includes at the same time the intercostal muscles. The two layers of stitches are solidly united, thus preventing the penetration of air through the weak intercostal closing. A third layer of sutures reconstitutes the trapezius. Thus the functional disturbances as well as the muscular trauma have been reduced to a minimum. Drainage is not necessary. We do not drain the "pocket," for coughing may expel the air and the lung thus become readherent.

The Anterior Approach.—To collapse lesions situated anteriorly we have proposed and elaborated a technique of P.A.E. by the anterior approach. The various phases of the operation are similar to those of the P.A.E. by the posterior approach. Following upon an horizontal incision, the pectoralis major is separated without being sectioned. Ten to 12 cm. of the second rib are easily resected. It

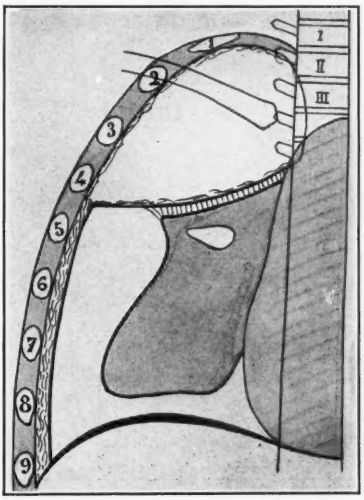

Fig. 5.—Combined intra- and extrapleural pneumothorax. Although the inferior limit of the surgical collapse extended far below the limits of the adhesions, there still exists a veritable horizontal partition of pleura which supports, crownlike, the apex of the lung. The cavity is drawn out as though by an adhesive band.

is difficult to scrape the pericondrium. This, however, must be done with the greatest care for the internal mammary vessels lie just behind. In addition it is necessary to remove the second costal cartilage to permit the detachment of the anterior costomediastinal cul-de-sac of the aortic arch and of the large ascending branches. By the posterior approach the freeing of the anterior cul-de-sac of the pleura is impossible.

Extent of the Décollement.—The extent of the décollement varies with the case. Its inferior limit should extend beyond the main lesion. One has always to take into consideration the fact that a certain amount of reattachment frequently takes place and that the volume of the pocket always diminishes somewhat in the weeks that follow the operation. The x-ray picture will help determine the point of cleavage; the pleura should be detached for about two intercostal spaces beyond the inferior projection of the lesion. To secure a long lasting P.A.E. the pleura should be firmly detached.

The effective collapse by the posterior approach rarely goes beyond the seventh rib posteriorly, and the inferior border of the first costal cartilage anteriorly. The pulmonary apex, even though freed, remains attached to the tracheal bifurcation. It is impossible to collapse it sufficiently to detach its anterior aspect under the control of the eye. This is equally true of the anterior approach; the effective collapse descends easily to the inferior border of the third costal cartilage but does not go beyond the fifth or sixth rib posteriorly.

Frequently one is misled by a false sensation of having freed the pleura. The freed pleura caves in on itself and fixes on to the unfreed pleura, thus the exploring finger does not feel the slight thickness of the pleura folded in on itself, and one has the impression of having collapsed to a degree farther down than one has in reality. It is thus necessary to see, under the control of the eye, what has been done, so as to be sure that the zone in question is entirely free.

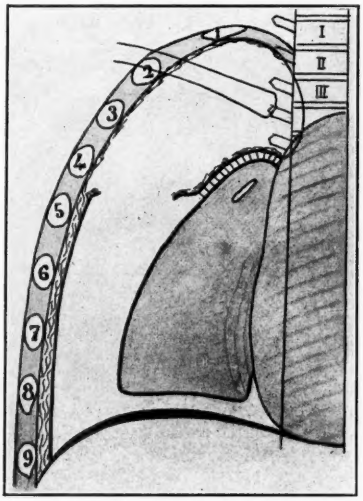

Fig. 6.—Mixed pneumothorax. The pleura has been sectioned all around and at a distance from the lung. The result is the same as with the ordinary pneumothorax. The only difference is that the apex is covered by the parietal pleura.

POSTOPERATIVE SEQUELAE

The operation involves a minimum risk of shock. Postoperative pain is slight and of brief duration, especially if the wall has only been separated and not cut.

The temperature rises very little; in an afebrile patient before the operation the temperature remains frequently below 38.5° C.

However, it is a curious fact that in cases which appear the easiest and allow of a rapid detachment one sometimes observes rather severe shock with pallor, tachycardia, and temperature of 40° C.

The lung, once freed, collapses. Roentgenograms made immediately after the operation show a decrease in the volume of the pulmonary apex which, however, conserves its shape. This picture persists in the P. A. E.

even where the extent of the detachment is considerable. Most frequently the pocket is of medium size and ovoid in shape. The retracted lung shows a shallow depression. The lower limit of the P. A. E. becomes adherent at its periphery, and the floor of the pocket, i.e., the apex of the lung, becomes concave.

Spontaneous Extension of the Detachment.—Sometimes, when at the end of the operation the finger has felt and the eye seen that the inferior limit of the detachment extends, for example, to the sixth rib posteriorly, to the fourth laterally, and to the second anteriorly, the x-ray picture, the next day, shows the extrapleural pocket extending much farther down—to the ninth rib, for example, or even as far down as the diaphragm. The picture is exactly that of a total pneumothorax of the pleural cavity. The extrapleural detachment has thus completed itself spontaneously and a collapse greater than intended has been obtained.

Immediate Re-expansion.—Twice we had the disagreeable surprise of observing the complete re-expansion of the apex during the night that followed the institution of the P. A. E. The roentgen film taken the day after the operation was identical with that which preceded the operation. The intercostal sutures may perhaps be held responsible for this. It is probable, however, that coughing expelled the air from the detached cavity into the mediastinum. Perhaps, also, when the detachment offers no difficulty, as in the case of a recent tuberculosis, the subpleural tissue may possess a special absorbing capacity.

Here, as elsewhere, in the last analysis one must incriminate the absence of retractile capacity on the part of the lesion. We have had the occasion to free such a lung that had no tendency to retract spontaneously. Is it not well known that we can only help the natural processes of nature to take place and that we cannot go against them?

MAINTENANCE OF THE EXTRAPLEURAL PNEUMOTHORAX

It is a delicate matter to maintain the pneumatic pocket. It requires constant supervision by means of x-ray examination and the check-up of the pressure in the pocket. Fluoroscopic examinations should be made the day after the operation and repeated a number of times during the week following. Special attention should be given to the volume, the limits and variations of the pocket, the shape of the apex, the tracheomediastinal displacement toward the unaffected side, and the quantity of fluid. In the early period of the pneumothorax the pressure in the detached pocket may increase or decrease considerably within a short time. Later, these changes in the pressure give way to a more stable equilibrium.

An exploratory puncture will likewise permit the injection into the pocket of a small amount of lipiodol. We inject, generally, 10 c.c. of a 40 per cent solution of heavy lipiodol which marks the inferior limit of

the detachment, and 10 c.c. of a 0.5 to 1 per cent solution of light lipiodol, which floats on the surface of the serosanguineous effusion and covers the apex with an opaque layer. Greater details can thus be more clearly brought out.

Lipiodol probably also prevents adhesions from forming, and acts as a coating on the subpleural cellular tissue.

The role of the physician consists essentially in warding off the accidents of increased or decreased pressure. In the event of increased pressure a decompression is effected by removing air or fluid. In the event of decreased pressure a small quantity of lipiodol and air is injected. Care should be taken against too great refills: 50 to 75 c.c. suffice usually to bring the pressure up to zero. Later even small quantities of air may bring the pressure up to plus 2 or plus 4. This is not a disadvantage. However, establishing a positive pressure from the very onset of the P. A. E. is poor technique, for the cellular spaces are permeable and the insufflated air will merely filter into the mediastinum and the neck, thus creating an emphysema.

After the fourth day the only eventuality is the progressive reattachment of the pocket. In this case there is no disadvantage in increasing the pressure or even in preventing the reattachment by means of an extrapleural oleothorax.

When the operative suture has become airtight, the pressure can be increased to plus 8, plus 10, plus 15, for we are not dealing here with a serous membrane as sensitive as that of the pleural cavity. Even very high pressures can be tolerated without danger. We have never observed any accident due to increased pressure established under these conditions. Experience has taught us, however, that high pressures are not more useful nor do they last longer. To pretend that they prevent adhesion is to reason somewhat naïvely. In reality a high positive pressure does not last as such from one insufflation to another. In general we maintain a pressure that varies between plus 2 and plus 6.

Characteristic of the refills that maintain a P. A. E. is the rapidity with which positive pressures are established with even small quantities of air. Frequently, 75 to 100 c.c. of air will raise the pressure from minus 5 to plus 12, plus 15, plus 20.

During the first ten to fifteen days, the refills should be repeated at very short intervals. Later, however, the intervals can be lengthened (one to two months), because the subpleural tissue does not possess the same capacity of absorption as the external aspect of the lung.

An effusion appears immediately after the operation. Usually it consists of a sero-sanguineous fluid and when tapped the third or fourth day it does not recur. At other times the effusion is predominantly sanguineous; the clots occupy the lower part of the collapsed area.

We believe that it is preferable not to let a slowly progressing effusion fill the whole extrapleural cavity. It has been said that it acts as a hemothorax preventing adhesion. This is very doubtful because the effusion is slowly resorbed and the pocket diminishes proportionately. If it be necessary to create a block against a possible symphysis we prefer the injection of lipiodol.

Progressive Readhesion.—In the event of a progressive readhesion the pocket can be blocked off with oil.

The extrapleural oleothorax should not be performed in a one-stage operation or performed without preparation. The first postoperative injection of lipiodol will give an indication as to the sensitivity of the walls of the air pocket. If lipiodol provokes a state of intolerance, the injections can be continued with gomenol.

Oleothorax is instituted by injecting 100 to 150 gm. of oil and repeating the injections at intervals of eight to ten days. This technique permits the progressive displacement of the air of the P. A. E. by a nonabsorbable oil without provoking important static disturbances in the wall of the pocket (lung and mediastinum). To fill the pneumothorax with oil without changing the pressure, one has only to place the patient in decubitus and puncture the pocket at its culminating point. Oil is injected and air withdrawn alternately.

Should a symphysis prove irreducible thoracoplasty may be indicated.

COMPLICATIONS

The immediate postoperative accidents may be grouped as those due to a decrease or an increase in pressure.

1. The *decrease of pressure* has one serious consequence: the reattachment of the apex. Should the apicolysed pocket diminish and the apex tend to re-expand, an urgent insufflation is indicated to prevent symphysis.

2. The accidents due to an *increase in pressure* are more numerous. Clinical symptoms are clear: dyspnea, a sensation of being suffocated, and distress. Cardiac symptoms, pallor, and cyanosis, occasionally accompany the respiratory difficulty. In operating on the right side, obstruction of the return circulation may occur.

Roentgenograms show the mediastinum drawn over to the unaffected side; the trachea and heart are displaced by the pocket. The inferior pole of this pocket depresses largely the lung beneath. Tapping relieves the patient immediately.

3. Occasionally *emphysema* occurs. Subcutaneous emphysema is generally an epiphenomenon of no importance. However, excessive refills of air performed before the fifth day may lead to dramatic emphysematous accidents such as abundant subcutaneous and mediastinal infiltration with compression of the veins of the base of the heart.

Coughing, in certain cases, may provoke intermittent increases in pressure, which explains how collections of air or fluid form between the superficial muscular layers. Radiograms and clinical signs show that these cavities communicate in collar button fashion with the P. A. E. This complication may lead to fistulas of the skin. These accidents, due to nonairtight sutures, show how important is the careful closing of the wound in several layers. All around the parietal breach the first muscular layer should be solidly united to the intercostal muscles.

4. *Hemorrhages* into the collapsed pocket may occasionally occur. They are often the results of rupture of small mediastinal or intercostal veins of the subpleural tissue, caused in the course of the collapse.

The vessels of the pleurae are derived from the intercostal veins and arteries. Small arterioles given off along the intercostal spaces traverse the endothoracic fascia and run to the pleurae. They are of such small caliber that in the normal patient cleavage is usually avascular. However, they may bleed if congestive peripleuritis exists or if the adhesions are of recent date and still inflammatory and vascularized.

5. *Perforations* of the cavity into the extrapleural pocket may take place in three ways: (1) Accidental operative perforations may be brought about in the course of forcing an adhesion. Superficial cavities require extreme care. (2) Postoperative perforations may result from necrosis of the external wall of the cavity. It is known that the external wall of superficial cavities, especially of large ones, receives its nutrient or trophic arteries from the intercostal arteries. They are newly formed vessels of cicatricial tissue. They run directly from the thoracic wall to the lung, traversing the adherent pleurae and the endothoracic fascia. Collapse, in separating the lesion from the area which supplies its vascularization, causes a massive necrosis of the wall of the cavity.

The most frequent perforations are those due to the extension of the pathologic process. Caseation progresses centrifugally and causes necrosis of the superficial wall of the cavity. This is likewise the pathogenesis of most perforations in P. A. I. Irritation of the sympathetic nerves in the course of the operation is probably an important factor in determining these exacerbations. These perforations infect the collapsed pocket and a parietal fistula is thus established (the communication is, therefore: bronchial—extrapleural—cutaneous).

6. *Suppuration* of the postoperative effusion may occasionally occur. Of course, ordinary pyogenic suppuration of ex- or endogenous origin may occur. Most interesting, however, are those which appear to be aseptic, although the presence of lymphocytes and tuberculization of the guinea pig indicate the tuberculous nature of these suppurations.

Certain investigations which we have pursued for a number of years have aided us in tracing the nature and origin of these tuberculous effusions unaccompanied by perforation.

Frequently, in the course of extraperiosteal thoracoplasties, ganglia and relay nodules were removed from the intercostal spaces or from the endothoracic fascia near the neck of the ribs. Even in purely "pulmonary" cases, where no recent nor long standing pleural inflammation could be demonstrated, histologic examination of these nodules almost always showed tuberculous lesions. It is easily conceivable how the opening of the lymphatic spaces may inoculate the collapsed pocket. It is surprising, however, how these effusions will frequently yield to a few punctures and lavages, and the P. A. E. can then be continued as before. These factors, however, do not adequately explain the pathogenesis and behavior of effusions in the course of a P. A. E.

The facts that these effusions are frequent and of a relatively benign nature and have a rapid course, that macroscopic tuberculous lesions in the walls of the collapsed pocket are absent, and, finally, that strictly aseptic puriform effusions exist, show that we are not dealing with an ordinary type of inoculation of the wound. A strict microbic theory does not satisfactorily explain the phenomena.

The nervous action of the sympathetic system certainly plays a role, either by inhibiting the automatic permanent defense of the pleura against infection, thus permitting the development of a purulent pleurisy, or by causing the formation of an aseptic puriform effusion, or, lastly, by provoking in one way or another an exacerbation of the tuberculous process. These problems are the subject of our present investigations.

Claude Bernard opened the way to the study of these problems. By stimulating the superior cervical ganglion in the cat he invariably determined the appearance of a purulent pleurisy. The cat, doubtless, is particularly sensitive in this respect. However, the hypothesis of a similar mechanism in man is not, a priori, absurd.

Of all surgical operations on the ribs and pleurae, the P. A. E. is the one which most affects the sympathetic nerves. In the course of collapsing the apex and the mediastinum, the fingers, the compressor or the "collapser" constantly irritate the stellate ganglion, the cardiac nerves, and the pleural nerve fibers.

Focal reactions.—Focal reactions may appear as a result of even the most cautious handling of the lung and the inferior cervical ganglion. The reactions are more frequent and serious after operation for young or poorly stabilized lesions and vary from slight exacerbation of short duration to the formation of an important infiltration or of a large atelectatic zone in the neighborhood of the lesion. These accidents are not confined to the extrapleural pneumothorax, they appear to be even less frequent after P. A. E. than after thoracoplasty. My teacher Rolland gave a complete and masterly description of them some years ago.

IMMEDIATE RESULTS

A lesion well collapsed is usually followed by a rapid, progressive, and important improvement. Clinically, the cessation of the expectoration, the disappearance of toxic symptoms, and gain in weight are evident. The earlier the lesion is treated, the more rapidly will the sputum become sterile and tend to disappear altogether.

As regards the sputum, the effects of a P. A. E. are similar to those of a P. A. I. We have observed the disappearance of bacilli from the sputum in three to six weeks. Several months after our first P. A. E. the sputum has still remained free of tubercle bacilli. Naturally, sufficient time has not yet elapsed to allow of a final judgment. In pulmonary tuberculosis Time does not respect whatever may have been done without due consideration for its effects.

INDICATIONS

Thus the indications cannot as yet be determined with precision. A few years must elapse since our early operations before one can judge the action of a P. A. E., and even then it will require several years before this technique receives the double sanction of time and number.

However, contrary to opinions expressed in publications up to this date, we believe that the indications are numerous and that the P. A. E. is frequently an operation of election.

In view of the fact that the P. A. E. can be done in cases with moderately active bilateral lesions, it would seem that the P. A. E. holds out many possibilities. Cavity lesions that constitute indications for P. A. E. are rather numerous, but they must fulfill a number of conditions which we shall enumerate. If these conditions are not satisfied, the danger of accidents is considerably increased. The future may reveal more extensive indications; however, we have not yet reached that point.

The indications for surgical treatment of pulmonary tuberculosis should be considered from two points of view: the anatomoclinical form of the disease, and its potential development.

Indications Considered from the Anatomoclinical Point of View.

1. The lesion must be well localized. A slight infiltration in the tissue surrounding the cavity is not a contraindication, but extensive, dense, ulcerocaseous or fibrocaseous lesions are distinctly contraindicated.

2. Only apical or subapical lesions can be thus collapsed. The reasonable lower limit of the lesions to be collapsed seems, at the present time, to be the sixth or, if absolutely necessary, the seventh rib posteriorly. The effective collapse rarely goes below the eighth rib posteriorly, and the second rib anteriorly (we have discussed above the technical possibilities of apicolysis in anterior and posterior approaches).

3. Small central cavities constitute the best indication. It is with such lesions that the method gives best results and rapid improvement, comparable to the results of the most successful P. A. I.

Cavities situated centrally and posteriorly, or centrally and anteriorly, as well as lesions of the costoclavicular angle may be operated on, but subcortical lesions may not.

The importance of exactly determining the situation of the lesions cannot be overemphasized. Detailed fluoroscopy and x-ray films in the oblique position are indispensable. Tomography is likewise of the greatest help.

Indications Considered From the Clinical Point of View

1. *Age.*—Naturally, early lesions respond better than others. They close rapidly and completely. We have, however, seen cavities three to four years old collapse completely and rapidly by P. A. E. contrary to all expectations. In the case of early lesions, even successfully operated on, the prognosis must be held in suspense for a long time. The same holds true for the P. A. I. In the early stage of a lesion, its future course is always an unknown quantity.

2. *Course.*—Up to the present our operations were performed for slightly active or quiescent lesions. In these cases, postoperative symptoms were insignificant and unaccompanied by fever. We have, however, also operated on active lesions in patients with relatively good natural defense. Although the postoperative symptoms were occasionally rather dramatic, it has seemed to us that the P. A. E. was an excellent operation in these cases. The P. A. E. is, of course, contraindicated in patients with very active, acute lesions (these patients respond equally as poorly to the P. A. I.). However, between the "galloping" form, which is beyond all therapy, and the chronic, torpid forms accessible to thoracoplasty, there exist a great number of patients with moderately active, poorly stabilized lesions (for whom thoracoplasty thus constitutes a danger), and where natural defenses are relatively good. If the pleural cavity is free a P. A. I. is indicated. In the past, before undertaking a thoracoplasty, it was customary to wait until the lesions were stabilized. Today, however, the P. A. E. is the operation of choice in these cases.

To sum up, now for the first time (and aside from phrenicectomy) surgical treatment of pulmonary tuberculosis is in possession of a method of treating active, acute lesions (*à chaud*), thoracoplasty being the operation for chronic, quiescent forms (*à froid*).

3. *The P. A. E. for Children.*—The P. A. E. can be performed in children. My teacher, M. Maurer, assisted by Dr. Lowys successfully performed a P. A. E. in two children.

4. *During pregnancy* when the operation must be done rapidly and with the least possible shock, the P. A. E. is likewise indicated.

5. P. A. E. may be done in the presence of *bilateral lesions,* since the operative traumatism is reduced to a minimum. Schmidt performed a number of P. A. E. in these forms with satisfactory results.

6. An elective localized collapse may be executed in certain *lesions of the base* of the lung.

7. The P. A. E. may be performed in *two stages.* In the first stage a small apical pocket is made. In the second stage the collapse of the base of the lung is completed after resection of the sixth rib.

CONTRAINDICATIONS

The contraindications follow from the explanations that we have given above and are conditioned by (1) the situation of the cavity and (2) the course of the disease. These are:

1. Extensive lesions with diffuse caseous infiltrations, highly organized lesions, fibroid lobitis, rigid forms, and cases in which honeycomb-like lesions are buried in a mass of fibroid tissue.

2. Voluminous cavities and especially superficial, subcortical cavities (the dangers of operating subcortical lesions whose walls are weak and poorly vascularized have been mentioned above).

3. Lesions of long standing surrounded by a rigid shell (although it is always a difficult matter to determine in advance whether a cavity will collapse or not).

4. Diffuse granular lesions, the macronodular forms, and those serious forms which follow an acute course.

5. Lesions accompanied by, or that were accompanied by, purulent pleuritis, or merely a doubtful fluid.

6. The absence of signs of retractibility indicates a bad operative prognosis, at least insofar as clinical lesions are concerned. Lungs which do not tend to retract can no more be cured by P. A. E. than by thoracoplasty.

THE PLACE OF EXTRAPLEURAL PNEUMOTHORAX IN COLLAPSE THERAPY

A Comparison Between the Extrapleural Pneumothorax and the Usual (Intrapleural) Pneumothorax

Respective Indications of These Two Operations.—The common intrapleural pneumothorax is still the method of choice for treating tuberculous lesions. The P. A. E. should not be undertaken unless the P. A. I. is impossible or ineffective. When the pleura is completely adherent, the need for a more active therapy arises; it is here that the extrapleural pneumothorax finds its indication.

One of the greatest dangers to which many tuberculous patients are exposed is the possibility of having unsuccessful artificial pneumothorax for a considerable length of time, despite the persistence of apical ad-

hesions. The matter is of such importance that we do not hesitate to repeat what is well known to all, namely, *artificial pneumothorax which does not collapse the cavity of an adherent apex should not be continued.* And the exceptions merely confirm the rule.

A pneumothorax that provokes complications (perforations, purulent pleurisy) is an unsuccessful pneumothorax.

Surgery, resorted to in time, has so amply demonstrated its usefulness in curing and saving the lives of patients that in the event an artificial pneumothorax would fail to collapse a cavity or suppress positive sputum within one to two months after its induction, one should not hesitate to consult the surgeon as to the possibilities of a surgical collapse. In the past this hesitation to proceed to thoracoplasty in patients with a recently performed pneumothorax (even though the chances for the success of the artificial pneumothorax were slight) was quite understandable. The patient naturally hesitated when advised to accept so permanently mutilating and so formidable an operation as thoracoplasty. Now, however, with the advent of P. A. E. it should be easier to persuade the patient to accept a simple apicolysis which either supplements the intrapleural pneumothorax or, when substituted for it, produces the same results.

Mixed Pneumothorax.—A pneumothorax that has not separated the upper lobe adhesions can be transformed into a perfectly satisfactory collapse. First, the apex is detached as in the usual P. A. E. When the adherent lung area is freed, the pleural cavity is opened and the pleura is sectioned crown-wise along the thoracic wall (likewise anteriorly and posteriorly, though this is more difficult). The pleural cavity thus communicates largely with the collapsed area. The freeing of the lung is thus complete and, technically, the pneumothorax is satisfactory.

This mixed pneumothorax (intra- and extrapleural) can be performed in one or two stages. In the one-stage operation the cleavage of the apex and the sectioning of the pleura are done in one stage. In the two-stage operation an extrapleural pneumothorax is first established and remains separated from the intrapleural pneumothorax by the detached parietal pleura. In the second stage the pleura is sectioned in the neighborhood of, as well as at a distance from, the lung. In general there is no point to letting the two pockets (the P. A. E. and the P. A. I.) coexist side by side, for at the limit of the collapsed zone the pleura forms a sort of diaphragm-like band that frequently keeps the cavity open.

Extrapleural pneumothorax is indicated in three groups of cases: (1) When the pleura is adherent and there exists a small quiescent cavity in the center of the apex; this is the indication of choice. (2) When a pneumothorax already exists but the apex is adherent and the lesion stabilized. By sectioning the pleura along the thoracic wall the attempt is made to collapse the apex and unite the two pockets.

3. When moderately active lesions are involved in those cases analogous to the preceding ones. These are less certain indications, but cautiously operated, however, the results are frequently very satisfactory.

To sum up, when pneumothorax becomes impossible or proves unsuccessful, one should keep in mind the possibility of performing an extrapleural pneumothorax.

Extrapleural Pneumothorax and Thoracoplasty.—Judging from present day trends, the indications of P. A. E. appear to be fewer than those of P. A. I. (given similar lesions and an adherent pleura). However, the indications for P. A. E. are more numerous than those for thoracoplasty.

The advantages of the method are many; it is a relatively benign operation, it can be done in one stage, and the collapse is complete. However, as compared with thoracoplasty, an important disadvantage of the method lies in the necessity of refills over a long period of time with all the uncertainties that this entails. Thoracoplasty once terminated merely requires close supervision and a prolonged stay in a sanatorium, whereas the maintenance of a P. A. E. entails repeated punctures, refilling, and injections as in the intrapleural pneumothorax.

Another decided advantage of the thoracoplasty is the permanence of the results. When thoracoplasty is followed by the disappearance of cavity and expectoration and when repeated analyses show negative sputum, the probabilities are great that the results will last. Recurrences are indeed very rare. Not until at least ten years more have passed will it be possible to compare the results of extrapleural pneumothorax with those of thoracoplasty.

When collapse proves impossible, what are the alternative measures that can be taken?

(a) In certain patients with good natural defense and where the lesion is not active, the P. A. E. may be the operation of choice. However, in these cases the technical conditions of a thoracoplasty are likewise fulfilled. Should their cleavage prove to be impossible, the procedure must be changed and one must resort to rib resection.

(b) In other patients, with poor natural defenses, and in whom the lesions are active, a P. A. E. may be undertaken, for thoracoplasty is dangerous. Should the cleavage prove impossible, the only thing left to do is to close the operative wound.

It is important in this case to determine in advance whether thoracoplasty can be done, or whether the wound must forthwith be closed. In the first case, a Maurer incision is made. In the second case, our oblique straight line incision should be employed. Besides, a P. A. E. may stabilize a lesion and thus prepare the way for a future thoracoplasty.

We should like to call attention to the fact that in the establishment of a P. A. E. not only is it difficult to decide upon the operation, but the surgical technique is a most delicate one. The margin of security in the P. A. E. is small. This should always be kept in mind before attribut-

ing to the method of P. A. E. failures which may result from too vague indications or from a lack of rigorous and cautious technique.

One foresees great possibilities for P. A. E. It is indicated, first of all, in a large number of cases where the lesions are too young and too poorly stabilized to permit a thoracoplasty. Then, too, P. A. E. may frequently be used in lesions for which hitherto thoracoplasty seemed the only feasible intervention. Lastly, when the moment for thoracoplasty has passed because of lessened resistance or bilateral lesions, there still remain many cases which can benefit from a P. A. E., and even after an inadequate thoracoplasty a P. A. E. may still be effective. In return, acting as a preparatory or auxiliary operation, the P. A. E. will widen the field of indications for thoracoplasty.

CONCLUSION

The P. A. E. is an operation of choice for a number of reasons: it is a less traumatizing intervention than thoracoplasty; it can be done in a one-stage operation; it is less painful and more easily tolerated; its action approximates mostly that of P. A. I. which is the most physiologic intervention of the lungs.

The P. A. E. may be used in urgent cases because its action is frequently as rapid as that of a P. A. I. In pregnancy, for example, when one must act quickly, the P. A. E. is indicated. The action of phrenicectomy, in this case, would be too slow.

It is essentially a *conservative* operation for, given similar lesions, it permits a more selective collapse than does the most conservative thoracoplasty.

It is the operation of necessity in advanced cases where the choice of treatment is limited and thoracoplasty cannot be undertaken.

The fact that extrapleural pneumothorax may be an operation of choice, that is, can be used in urgent cases, and the possibility in certain cases of its being the operation of necessity, makes of it a procedure with immense possibilities—unless the results in the future should prove the contrary.

Addendum.—This article was written in October, 1937. The experience of the year 1938 has not suggested any modifications of what we have written here. On the contrary, we are more and more of the opinion that the P.A.E. should be employed with prudence, but sooner. Many authors in France and abroad do not share this view.

The best indication for the P.A.E. is the recent and discretely developed lesion with a small cavity and situated at a distance from the wall. A large cavity, situated near the cortex, is an absolute contraindication. Let us note that these cases are equally those which are the most favorable for P.A.I.

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EXAMINATION OF THE SPUTUM FOR MALIGNANT CELLS AND PARTICLES OF MALIGNANT GROWTH

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IN 1927 Dudgeon and Patrick[1] described a new technique for examining fresh tissues removed at operations. This technique has been in constant use at St. Thomas's Hospital ever since, and after the first thousand specimens had been studied a second report was prepared by Dudgeon and Barrett.[2]

In principle it consists of taking fresh tissues removed in the operating theater and scraping the surface of any abnormal areas with a scalpel so that a suspension of cells is obtained upon the blade; smears are then made upon glass slides and the preparations are fixed, stained, and mounted.

It has been established that, provided absolutely fresh tissues are used and that both fixation and staining are perfect, it is not difficult to diagnose pathologic lesions with the same accuracy as is possible with paraffin sections. In many cases the films are superior to the sections, since the cells can be studied individually.

When this work was first undertaken it was important to decide whether it was possible to recognize malignant cells when separated from their usual surroundings, for some pathologists have maintained that there is no criterion, apart from invasion, which would enable a person to say that a given group of cells is malignant. After studying a great number of smears the authors of these papers are convinced that there are characteristics of malignant cells which differentiate them from benign cells. It may be mentioned that the most striking features of malignancy are an irregularity in size, shape, and staining quality, which applies not only to the cytoplasm but also to the nuclei, and a tendency for the cells to separate from each other or to form syncytial masses in which the individual cell membranes are not discernible.

It has been stressed that in using this method absolutely fresh material is necessary because autolysis occurs soon after death and makes the cells unfit for examination, and so "it was feared that . . . the recognition of particles of malignant growth in the sputum would be impossible." Early attempts to apply this technique to material obtained from the urine, test meals, pleural fluids, and the sputum were disappointing, and the matter was not seriously investigated until Dudgeon made the following observation. He was asked to give an opinion about a patient who had recently had a cholecystectomy and whose convales-

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cence had been interrupted by the development of some postoperative pulmonary complication. The physical signs in the chest were indefinite, but there was some sputum which was watery in appearance and had fine streaks of blood in it. A specimen of sputum was taken for examination, and it contained many particles of columnar cell carcinoma (Fig. 1). The lesion in the gall bladder was a carcinoma and not a chronic cholecystitis, and the pulmonary symptoms were due to secondaries. Subsequent events confirmed this diagnosis because the patient died of the metastases in the liver and the lungs.

In view of this experience work was started four years ago by Dudgeon and Wrigley,[3] and large numbers of specimens of sputum from patients with different kinds of pulmonary disease have now been examined. They were soon able to publish the results of their work in the first hundred cases diagnosed clinically as malignant disease of the lung, and these were summarized again by Dudgeon in 1936.[4] He quoted the following results:

Total number of cases investigated	102
1. Cases diagnosed as malignant growth of the lung in which fragments of new growth were found in the sputum	43
2. Suspected growths of lung, but no evidence of growth in the sputum: ultimately proved to be nonmalignant	19
3. Proved cases of malignant tumors of the larynx, but no growth in the sputum	13
4. Suspected malignant growth with sputum positive, but these cases ultimately proved to be of an inflammatory nature	2

It is worth mentioning that particles of malignant growth have often been found in pleural effusions in recent years but that although some positive findings have been obtained from test meals and specimens of urine, the results in these last two fields have not been as satisfactory as in the sputum.

The present paper deals with Dudgeon's material to date and attempts to indicate the practical application and importance of the method as far as it has been determined at St. Thomas's Hospital during the last four years.

TECHNIQUE

The sputum is best examined as soon as possible after it has been coughed up or collected during bronchoscopy; but good results may be obtained from specimens examined as much as twenty-four hours after expectoration because for some unexplained reason cells in the sputum do not autolyze as quickly as those of post-mortem material.

The sputum must be sent to the laboratory without the addition of any preservative, saline, or disinfectant. It is poured onto an unglazed porcelain tile to enable the mucus and froth to be separated from the purulent or bloodstained parts. Samples of materials are selected with

a platinum loop and spread upon slides which are immersed *immediately*, before they have dried, in Schaudinn's solution. The composition of this fixative is: absolute alcohol, 1 volume; saturated aqueous solution of mercuric chloride, 2 volumes; acetic acid, a few drops for every 100 c.c. of solution.

The best results are obtained by leaving the slides to fix for fifteen to twenty minutes; they are then transferred to the second solution which consists of methylated spirit to which a few drops of tincture of iodine have been added. The iodine converts the mercuric chloride of the first

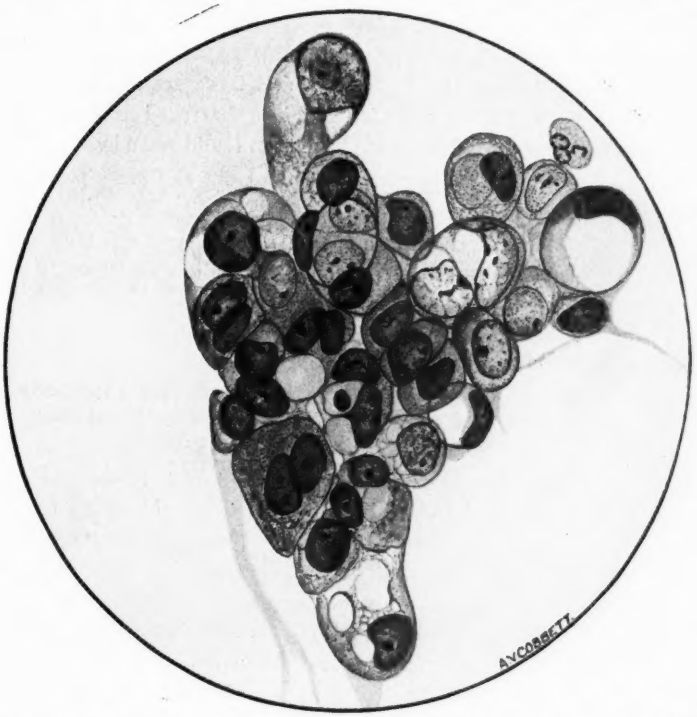

Fig. 1.—Columnar cell carcinoma in the sputum, secondary to a carcinoma of the gall bladder.

solution into the more soluble iodide so that when the slides are washed in distilled water all crystals are removed. In all this work with wet films Mayer's hemalum and eosin have been used as stains, and although many other combinations have been tried none have given such good results for routine examination. Dorothy Russell has, however, found it necessary to use special reagents for wet films of cerebral tumors.

Hemalum is made by adding 1 gm. of hematoxylin to 1000 c.c. of distilled water and heating until the hematoxylin has dissolved. To this solution 0.2 gm. of sodium iodate, 50 gm. of ammonia alum, and 20 c.c.

of glacial acetic acid are added. The dye improves as a nuclear stain as it matures and is best kept for a few weeks before use.

The slides are left in hemalum for about two minutes and are then washed for ten minutes in tap water until the color changes from purple to blue. The eosin should be a weak solution, and, after staining, the films are dehydrated with alcohols and xylol and mounted in the usual way with Canada balsam.

It is important to observe the following details of technique in order to produce specimens good enough to diagnose. An opinion should never be offered unless a perfect specimen is available, for poor films lead to incorrect diagnoses.

1. The "solid" parts and those in which there are streaks of blood should be selected, but no sputum should be discarded as unworthy of examination because malignant cells have been found when least expected.

2. Six or seven slides should be made from different parts of a specimen.

3. The more mucus there is in a specimen, the longer the films should be fixed because it is more difficult for the Schaudinn's solution to reach the cells.

4. Staining must be constant from specimen to specimen, because diagnosis is difficult if it is not. This applies particularly to the eosin which should not be too dark, and hence the advice that a weak solution should be used because a greater margin of error exists in one which takes three minutes than in one which works in fifteen seconds.

5. All films must be examined methodically since it is not unusual to find only one small group of malignant cells in a batch of six or seven slides. In some patients repeated specimens should be obtained, and in any case it is only positive evidence which is valuable.

6. The most likely places to find particles of tumor are at the edges of the cover slip and in patches where there are red blood cells.

7. It is advisable to search with a low-power objective and to examine suspicious areas only with the high power. It is practically always possible to pick up malignant cells in this way, and usually the diagnosis can be made from the low power alone. It is a useful working rule that doubtful groups of cells should not be regarded as malignant.

In practice it has been found that diagnosis by this technique is accurate and that errors are the result of inexperience rather than deficiencies in the method. At St. Thomas's Hospital a technician has mastered the details and is well able to mark all suspicious areas, without overlooking positive cases, for confirmation by a competent pathologist.

Malignant cells in the sputum are not always diagnosed by the same criteria as they are in films made from fresh tissues. These criteria hold good for cases of squamous cell carcinoma and for secondary growths;

but oat cell tumors which form the large majority of bronchial growths are diagnosed because one finds cells of special shape, size, and grouping which do not exist in a purulent sputum. These cells frequently do not look like malignant cells from other parts of the body, and it is only trial and error which have demonstrated their true nature.

THE CYTOLOGY OF SIMPLE PURULENT SPUTUM

Sputum contains masses of polymorphs which are often well preserved and are only really disintegrated in cases of lung abscess and suppurative bronchiectasis. These lie both in clumps and evenly throughout the

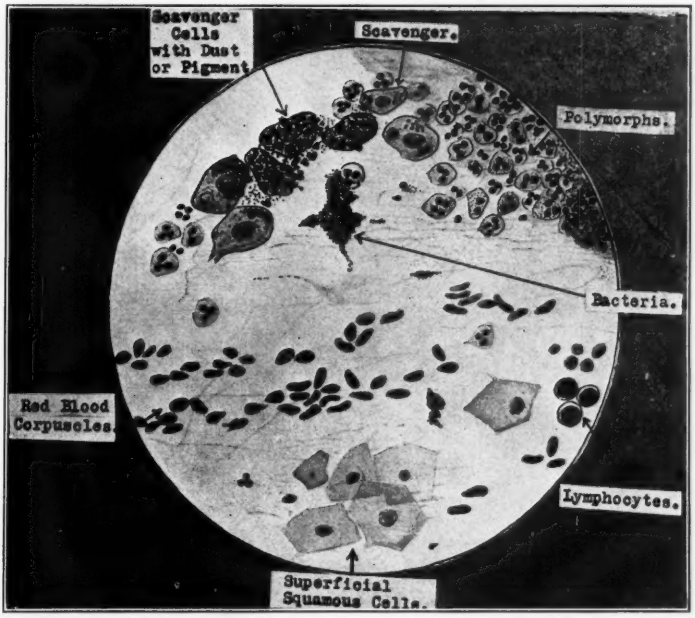

Fig. 2.—Diagram to illustrate various types of nonmalignant cells in purulent sputum.

whole film. In patients suffering from asthma the sputum contains large numbers of eosinophils.

Next in order of frequency are the superficial squamous cells from the mouth; they are easily distinguished because they are large, angular cells with small pale nuclei and clear cytoplasm which takes up the eosin, and they are quite different from the placards of squamous cells derived from the deeper layers of epithelium or from areas of metaplasia in the lungs. In many pathologic conditions such as bronchiectasis there may be large amounts of squamous epithelium lining peripheral bronchi and cystic spaces, and the sputum from such patients often contains cells from the palisade and prickle-cell layers.

"Dust cells" or "scavenger cells" are common in the sputum and are derived from the lining walls of the alveoli. They have been confused by some with malignant cells, but this mistake should not occur. They are large and assume a variety of shapes, and lie most commonly in clumps or streaks. The cytoplasm is abundant and sometimes vacuolated and it may contain large quantities of black or brown granules of dust or numerous red blood corpuscles, thus presenting the picture of "heart failure cells," or of granules which give the free iron reaction. The nucleus is small, relative to the size of the whole cell, and it

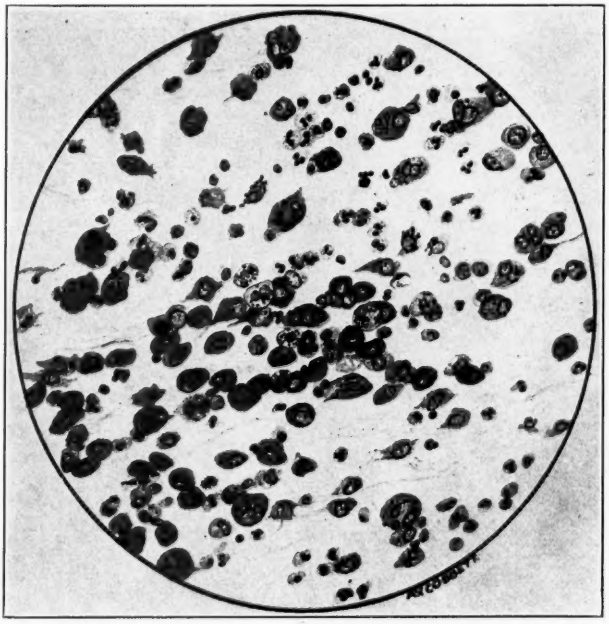

Fig. 3.—Typical "scavenger" cells. Note that these cells are separated from each other and not in plaques.

contains fine and delicate dots or rods of chromatin, as opposed to the more compact and dark material of malignant cells. Scavenger cells are isolated cells; they do not form placards but may appear to do so if closely clustered together.

The red blood corpuscles stain red and, owing to the fixation in Schaudinn's solution, are somewhat smaller and more elongated than in films made from fresh blood.

Lymphocytes are sometimes present in the sputum in enormous numbers, and it should not be difficult to distinguish them from oat cells. The nucleus of a lymphocyte is round, and the chromatin is arranged in a typical way, quite different from anything one sees in malignant

cells. This difficulty is overcome when one realizes that these cells can appear in the sputum and by using the high-power objective in doubtful cases. It is assumed that they are derived from the lymphoid tissue in the submucosa of the larger bronchi or that they have come from a hilar gland which has discharged into the bronchial tree.

Placards of normal bronchial epithelium are occasionally found. If one makes a film from the mucosa of a normal bronchus the majority of cells are perfect columnar ciliated epithelium, but in films of sputum from pathologic conditions ciliated columnar epithelium is rare; it is much more usual to find placards of epithelial cells which come from the deeper layers of the mucosa. The most difficult type of sputum one has to diagnose is that in which there are groups of both normal and malignant epithelium of this type.

The general background of films of sputum is mucus, debris, elastic fibers, and collections of bacteria.

MALIGNANT CELLS IN THE SPUTUM

The relative frequency with which different types of malignant growth have been found in the sputum in this series is shown in Table I.

TABLE I

		NUMBER OF CASES IN WHICH THE SPUTUM DIAGNOSIS WAS CONFIRMED BY SECTIONS MADE FROM POST-MORTEM MATERIAL	NUMBER OF CASES IN WHICH THE SPUTUM DIAGNOSIS WAS CONFIRMED BY SECTIONS MADE FROM MATERIAL REMOVED AT BRONCHOSCOPY OR FROM SECTIONS OF GLANDS
Oat cell carcinoma	55	19	11
Columnar cell carcinoma	37	12	15
Squamous cell carcinoma	18	6	5
Secondary carcinoma	9		

In two cases there were fragments of both oat cell and columnar cell carcinoma in the sputum and in only one has the diagnosis, as to the type of malignant cell, been different in the sputum and in the section obtained by bronchoscopy. In this case the sputum diagnosis was columnar cell and the section diagnosis was squamous cell carcinoma.

The high proportion of oat cell carcinoma and columnar cell carcinoma as compared with the squamous cell variety is interesting because the latter is the most common diagnosis in reports made upon sections of tumor removed through a bronchoscope.

Mr. F. J. S. Gowar, who has worked with this method at the London Hospital, tells me that in his material squamous cell carcinoma has been more common in the sputum than the other two varieties, and it is possible that the difference between his figures and these lies in the fact that he regards some cases as squamous which Dudgeon would call columnar.

In this series there has not been any chance as yet to examine sputum from a patient with an adenoma of the bronchus; but smears made from pieces of two such tumors removed by the bronchoscope were submitted to Dudgeon without details as to their origin, and he diagnosed carcinoma of a type similar to that seen in some papilliferous bladder growths. Without a great deal of experience it would probably not be possible to distinguish between adenoma and carcinoma of the bronchus. Nor is it possible at the moment to give any information as to whether cells from peripheral tumors of the lung, like the so-called "vascular endotheliomata" described recently by Tudor Edwards and Brian Taylor, could be found or recognized in the sputum. These tumors are rare, and their histology is difficult and varies from specimen to speci-

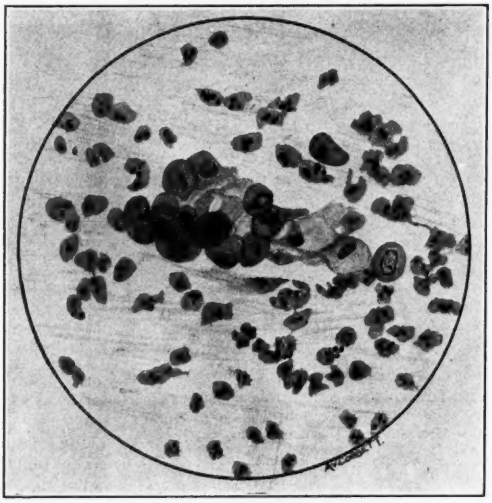

Fig. 4.—A plaque of cells in the sputum diagnosed by Dudgeon as an endothelioma. This patient died from endothelioma of the pleura.

men; they often appear to be circumscribed and almost encapsulated, and the development of secondaries is delayed. On the other hand the intermediate tumors of the lung, which arise from the smaller bronchi, beyond the point where they can be seen by the bronchoscope, have often been diagnosed by finding malignant cells in the sputum, and several cases of this type are quoted later on. The majority of tumors in this class are squamous carcinoma in type.

There were two patients who came to the Hospital with thoracic pain due to endothelioma of the pleura. In one the sputum was positive. This man developed a productive cough shortly before he died, and malignant cells were found. At the post-mortem examination no tumor could be seen in any of the bronchi, but paraffin sections taken from suspicious areas showed the malignant cells penetrating between the

cartilages of the bronchi into the submucosa, and somewhere they must have broken through. The whole lung, the pericardium, the hilum, and the great vessels were encased in the endotheliomatous tissues, and the post-mortem and histologic findings were in favor of a primary endothelioma of the pleura rather than a primary carcinoma of the bronchus with pleural secondaries. Cases of the latter type recorded in the literature have usually been oat cell carcinoma of the bronchus, but the cells in this man's sputum were very suggestive of endothelioma.

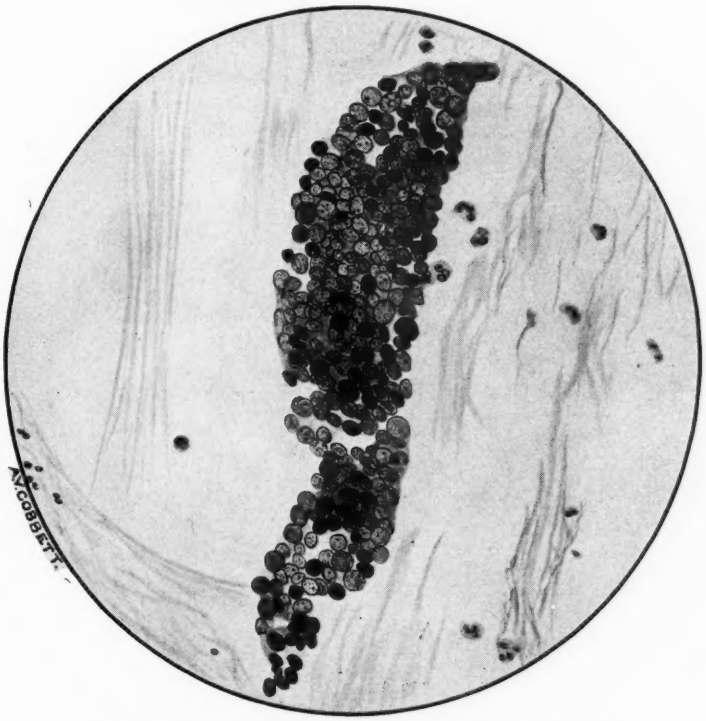

Fig. 5.—Large fragment of oat cell carcinoma in the sputum.

Oat cell carcinoma appears as groups of cells usually about 50 to 100 in number, arranged in streaks like the red blood cells, or in small clusters. The common findings are well shown in Figs. 5 and 6, in which the following points should be noted:

(1) The cytoplasm is scarce and inconspicuous. It forms a syncytium in those places where more than a few cells are huddled together, and may be absent towards the ends of a group. (2) The nuclei are all about the same size but are never properly round or oval like those of the lymphocytes with which they are likely to be confused. The

chromatin is fine and granular, and there are one or two tiny but obvious nucleoli.

Columnar cell tumors present a different picture, and it is these cells which have been confused with scavenger cells. Figs. 7 and 8 are drawings of typical specimens and have these characteristics: (1) The cytoplasm is plentiful and stains a bluish-purple color. It is granular and

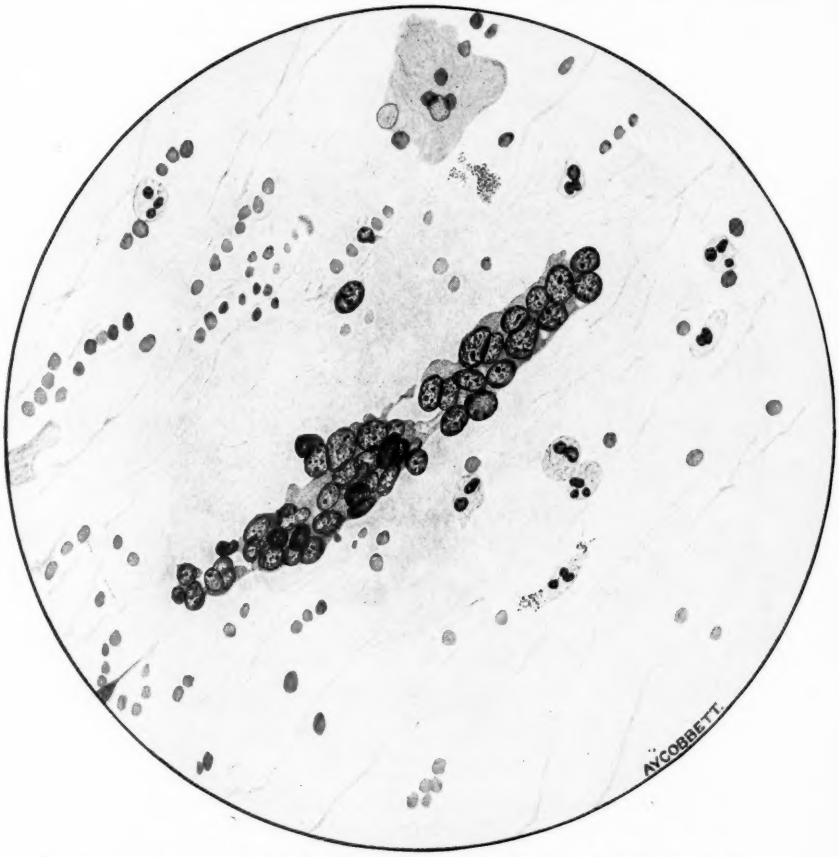

Fig. 6.—Another group of typical carcinoma cells of the oat cell type, but with more cytoplasm than in those of the last figure.

uneven and often contains inclusion bodies or vacuoles. The cell membranes may be poor or deficient and the formation of a syncytium is common. (2) The nuclei are large, irregular, and filled with coarse granules of chromatin; they vary enormously in size and shape. Secondary columnar carcinoma is difficult to distinguish from the primary growths described above, but in practice this difficulty does not often arise because the presence of the primary is usually known or sus-

pected, and the question at issue is whether the signs in the lungs are due to a spread or to other disease like chronic bronchitis.

In Dudgeon's series there have not been any cases of sarcoma in the sputum or of hypernephroma, but there is every reason to assume that secondaries of this type would be demonstrable occasionally because the positive cases of secondary carcinoma, which have been examined at autopsy, have not had any macroscopic connection with the bronchial tree.

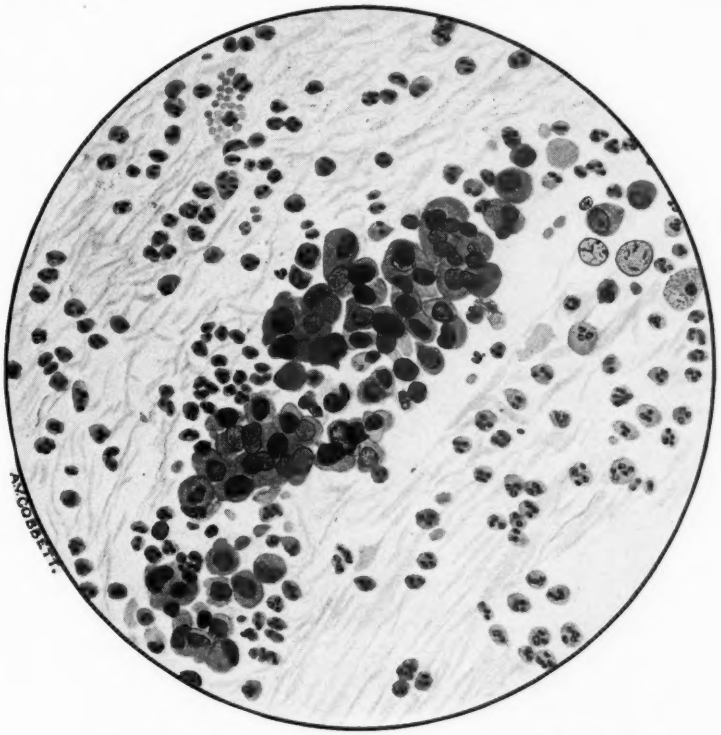

Fig. 7.—Fragments of primary columnar cell carcinoma in the sputum.

Primary squamous carcinoma is difficult to pick up and diagnose because the suspicious cells are few in number and often exist as isolated units. There is a lot of rather pink cytoplasm and a tendency, when two or three are gathered together, to form a little circlet rather suggestive of a whorl. The nuclei are large, irregular, and deeply stained, and sometimes there is definite keratinization.

CLINICAL VALUE OF THIS METHOD OF INVESTIGATION

It is proposed to illustrate the various ways in which this method of investigation has been useful at St. Thomas's Hospital by quoting the following brief case histories.

The first group illustrate that malignancy can sometimes be diagnosed from the sputum when other recognized methods, excepting exploration of the thorax, have been tried and failed. These are cases of carcinoma of the bronchi which cannot be detected with a bronchoscope.

CASE 1.—A patient, aged 69 years, came into the Hospital because he had noticed a heavy, tight feeling on the left side of his chest and because he had recently

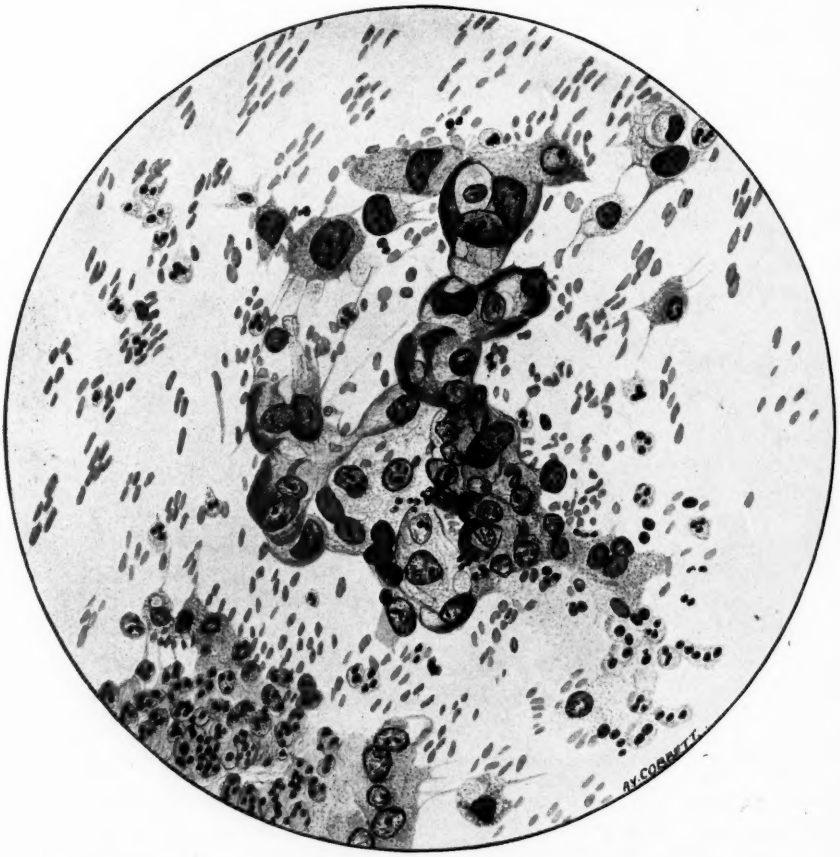

Fig. 8.—Papilliferous columnar cells from a case of primary carcinoma of the lung.

had two small hemoptyses. Two years before, a malignant ulcer had been removed from the lower lip and there was no local recurrence or cervical metastases. He stated that he had lost weight. The physical signs in the chest were those of bronchitis, a complaint from which he had suffered for many years. An x-ray film showed a hazy opacity about the size of a plum above and outside the left hilum; an artificial pneumothorax was induced and a new x-ray film demonstrated another opacity of the same quality in the left lower lobe. Malignant disease of

the lung was suspected, but there were no other physical signs. Bronchoscopy was negative. The sputum, which was mucoid, contained numerous collections of oat cell carcinoma.

CASE 2.—A man 52 years of age, who had always been perfectly fit, was admitted because he had developed a persistent cough and purulent sputum. Three weeks after the onset of these symptoms he became short of breath and noticed some blood in the sputum. A fortnight later he was admitted, and the only physical sign which was found was consolidation at left base. The x-ray showed an opacity involving the lower lobe on the affected side, and the radiologist reported that he was unable to say whether the lesion was an abscess or a growth. Bronchoscopy was done, but the diagnosis could not be made as the bronchi were normal. The sputum contained squamous carcinoma cells.

CASE 3.—A man aged 64 years, who had spent half his life amongst flocks of sheep in Persia, consulted his doctor because he had had a sudden and unexpected hemoptysis. He was admitted to another hospital where an x-ray film

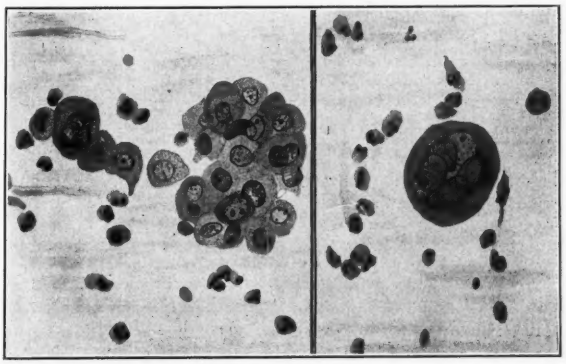

Fig. 9.—Drawings made from two cases in which squamous cell carcinoma was found in the sputum. The cytoplasm in these cells is well stained with eosin and unlike that seen in other types of malignant growth.

showed a sharply defined shadow the size of a small orange in the right upper lobe. A bronchoscopy was advised, and a stenosis of the right main bronchus was noted, but no evidence of growth was found. The diagnosis was not made as there were no other physical signs. His health improved with rest, and four months later he returned to work as a lambskin sorter. He then consulted his doctor again, because he had been advised that a further x-ray film should be taken to record the progress of the lesion. The x-ray showed no change from the previous film. At this point he consulted a chest specialist who diagnosed a hydatid cyst. The points in favor of this view were the fact that the shadow had not altered in four months (it was round, encapsulated, and cystic in appearance), the long association of the patient with sheep and dogs, and the fact that his brother had had an "exactly similar" lesion thirteen years previously and was still hale and hearty. The patient was convinced upon this point. He gradually became worse in health, and eleven months later he died in hospital.

The sputum was examined on several occasions, and oat cell carcinoma was found once. This patient probably had a peripheral lung tumor, and, although the sputum was never positive until the disease had advanced almost to its termination, it is interesting to note this evidence was ultimately available.

CASE 4.—An elderly man was admitted with a history of cough and debility. He had been unwell for thirteen months and came to the hospital because of increasing dyspnea on exertion and loss of weight. He had much purulent sputum which contained fragments of carcinoma. In view of this finding a course of deep x-ray treatment was advised by the physician but his condition was in no way improved by the treatment. A bronchoscopy was done to determine the site of the tumor in the hope that a radon applicator might be inserted, but unfortunately the growth could not be seen.

Three months later the patient died and the pathologist's report was as follows: "Bronchiectasis present at both bases; the bronchi were scarred and in places so denuded of epithelium that the cartilage was exposed." At the bronchoscopy this condition suggested submucous deposits of malignant growth, but sections taken at the post mortem proved that the tissue was cartilage surrounded by a great deal of chronic inflammation. "Throughout the lungs there were small globular patches of bronchopneumonia." Paraffin sections from these patches of so-called "bronchopneumonia" showed breaking down columnar cell carcinoma, and alveoli filled with inflammatory cells.

This case is particularly interesting because until the sections of the lungs were examined it was assumed that the sputum diagnosis had been wrong.

We have found that there are quite a considerable group of patients for whom the diagnosis is practically certain on clinical grounds and whose disease is far advanced. It does not seem justifiable to submit these to diagnostic procedures such as biopsies and bronchoscopies if the diagnosis can be proved from the sputum. One case will suffice to illustrate this point:

CASE A.—A chartered accountant, aged 59 years, became ill with shortness of breath and loss of weight. He developed other signs of bronchial carcinoma and at the end of six months was persuaded to consult a doctor. The sputum at this time was plentiful, purulent, and streaked with blood. He was emaciated and very dyspneic. Upon clinical examination there were firm and fixed glands in the neck and a pleural effusion on the left side. Two and one-half pints of straw colored fluid were aspirated, and after this x-ray films showed an abnormal shadow at the left hilum with glandular enlargement in the mediastinum. Apart from this the physical signs were negative. Actually this patient declined any other form of investigation, and it was fortunate that the diagnosis of carcinoma could be proved from the sputum.

Secondary deposits of growth in the lungs and pleura can often be proved in this way.

CASE B.—A man came to the surgical outpatient department complaining of an ulcer situated in the skin just below the left clavicle. Squamous cell carcinoma was suspected, but the possibility of a gumma was considered. A piece of the edge of the ulcer was taken for section; some blood and some sputum were also sent to the laboratory.

The sputum was reported upon immediately: it contained squamous cell carcinoma. The diagnosis was confirmed later by the other investigations.

CASE C.—A woman was operated upon for carcinoma of the left breast; there was no obvious glandular involvement, and a radical removal was done. Six years later she came to the follow-up clinic because of an aching pain in the left side

of the chest. Investigation showed a pleural effusion, which was clear and straw colored; x-ray films of the chest and spine were negative. There were no other physical signs but the pleural fluid contained many placards of malignant cells.

Another group of cases in which examination of the sputum has often been valuable is that of lung abscess. Malignant disease of the bronchus not infrequently produces an obstruction with suppuration and abscess formation distal to the block, and occasionally a tumor becomes excavated by central necrosis and is mistaken for an abscess. In view of the frequency of this association of abscess with growths, which some have maintained is as high as 30 per cent of all lung abscesses, it is valuable to have a method of diagnosis which can be used as well as bronchoscopy.

At St. Thomas's Hospital it has become a routine procedure to examine the sputum of all patients in whom there is any element of doubt in the diagnosis, and in such the pathologist is asked to look for malignant cells as well as tubercle bacilli.

Examination of the sputum for malignant cells should not be regarded as an alternative to the usual methods of investigation: it should be done in conjunction with these. The fact that it does not involve any discomfort to the patient enables it to be used not only for outpatient investigations but also for people who are ill and not fit for other procedures.

It is suggested that if the possibilities in this line were more widely appreciated and pathologists were prepared to devote the necessary time and practice to the diagnosis of malignant cells in the sputum, a certain number of early cases of carcinoma might be picked out by doctors who could obtain the sputum and send it to a pathologist before the patients were prepared to come to hospital for full investigation. In a recent Hunterian Lecture at the Royal College of Surgeons, Brock emphasized the fact that, by the time cases of carcinoma of the lung were submitted to him, practically all were beyond surgical treatment. A method which offers the possibility of improving early diagnosis in these cases is worthy of serious examination.

In preparing this paper I have had the helpful criticism and advice of Professor Dudgeon and I take this opportunity of offering him my thanks.

I have also to thank Mr. Walter Howarth for permission to use some of the illustrations which first appeared in Professor Dudgeon's article in the *Journal of Otology and Laryngology*.

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THE ACTION OF SULFANILAMIDE UPON THE GROWTH OF THE TUBERCLE BACILLUS IN VITRO*

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MONTREAL, QUE.

THE purpose of this investigation was to ascertain whether sulfanilamide exerts any effect upon the growth of the tubercle bacillus in vitro. This is a preliminary report. The results recorded here permit neither of conclusions nor of clinical application.

A nutrient egg agar medium previously described by one of us (A. G.) was employed in all experiments. This medium consists of a mixture of 0.8 per cent peptone, 1.7 per cent starch, 0.8 per cent agar, 3.5 per cent glycerin, 26 per cent egg, and gentian violet 1:30,000. The sulfanilamide dissolved in warm distilled water, was used in dilutions of 0.5 per cent and 1 per cent. It is well known that ordinary heating (100° C.) does not affect this drug. The quantity of sulfanilamide in the medium was estimated after inspissation and was found unchanged. In the concentration employed, gentian violet does not exert any appreciable bacteriostatic action upon the growth of the tubercle bacillus.

Sulfanilamide was added to the hot peptone-starch-agar solution; mixing permitted of even distribution of the drug. The drug agar mixture was then allowed to cool to 65° C. The glycerin-egg-dye mixture, previously filtered through five layers of gauze, was then added. The medium in its final form with sulfanilamide in combination was filtered through three layers of gauze, then tubed and slanted. Inspissation was carried out in the usual manner for two hours at 75° C. on the first day, and at the same temperature for one hour on the following day.

A fine suspension of a twenty-day-old culture of tubercle bacilli made up with a buffer solution hydrogen-ion concentration 8 (Clark and Lubs series) was filtered through two Whatman papers, No. 2. Two drops of this suspension were then seeded upon the slanted surface of the medium. A smear made of this seeded suspension revealed eight to ten bacilli per 100 power oil immersion field. In our first experiments the sulfanilamide was employed in the following concentrations: 1:100, 1:1000, 1:10,000, 1:25,000, and 1:100,000. After three weeks no growth was observed in the tubes with the 1:100 and 1:1000 concentrations of the sulfanilamide. There was some inhibition with sulfanilamide 1:10,000, but there were luxuriant growths with the two

*From the Department of Surgery, Jewish General Hospital, Montreal. This work was aided by a grant from Sally Ryan.
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weakest concentrations of the drug, namely, 1:25,000 and 1:100,000 (Fig. 1). The results were the same after six weeks with the exception that the medium with sulfanilamide 1:10,000 now showed heavier growths of tubercle bacilli (Fig. 2).

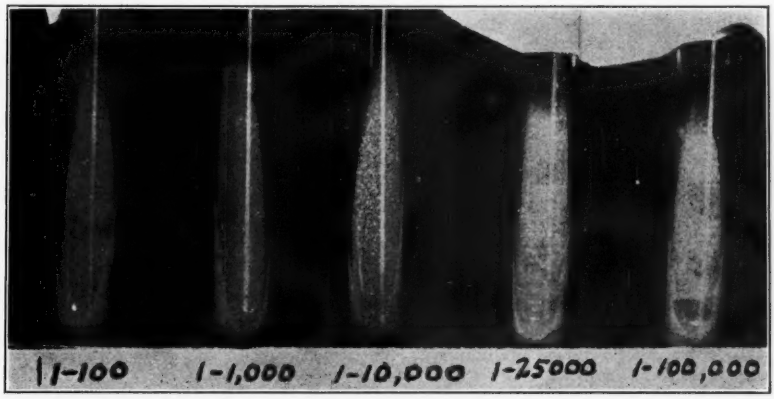

Fig. 1.—These tubes were seeded with two drops of a suspension showing eight to ten tubercle bacilli per 100 power oil immersion field. The concentrations of sulfanilamide have been indicated. There is no growth in the first two tubes (concentrations of 1:100 and 1:1000). Slight inhibition in the growth is to be observed with sulfanilamide 1:10,000. The photograph was taken after three weeks.

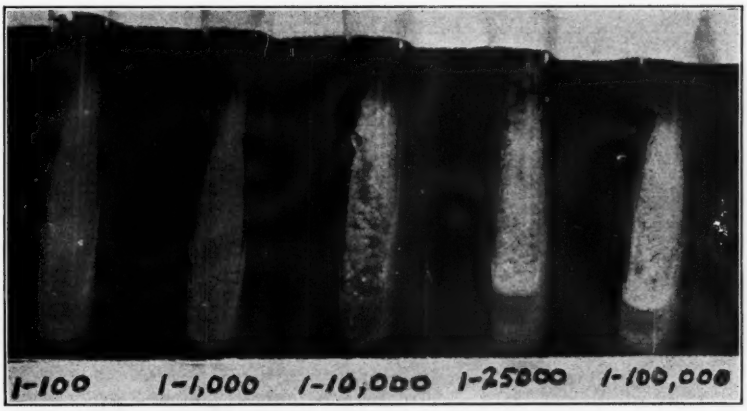

Fig. 2.—Same tubes as in Fig. 1, photographed after six weeks. Note the tube with sulfanilamide 1:10,000 now shows a heavier spreading growth. The first two tubes (1:100 and 1:1000) still fail to show any growth.

In a second series of similar experiments heavier suspensions of tubercle bacilli were employed. In this instance the suspension employed contained roughly 1000 more bacilli in the seeded suspension. No growth was observed with sulfanilamide 1:500 and 1:1000. Retarded growths were observed with a concentration of the drug of 1:5000 and to a very slight extent with 1:10,000 (Fig. 3).

In a third series of experiments two drops of a light suspension of tubercle bacilli which showed two to three bacilli per 100 power oil immersion field were seeded on the tubes. Clumps of bacilli were avoided as far as possible. Sulfanilamide was employed in the following con-

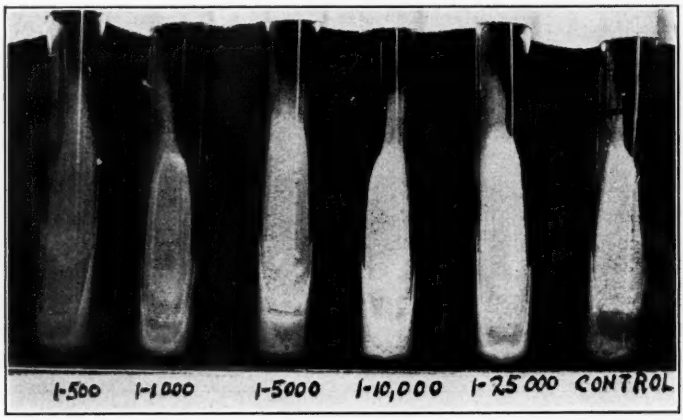

Fig. 3.—Tubes of the second series of inhibitory experiments after incubation for three weeks at 37° C. These tubes were seeded with a heavy suspension of tubercle bacilli. Note complete inhibition in the tubes containing sulfanilamide in concentrations of 1:500 and 1:1000; growth was retarded with sulfanilamide 1:5000 and to some slight extent with 1:10,000, early.

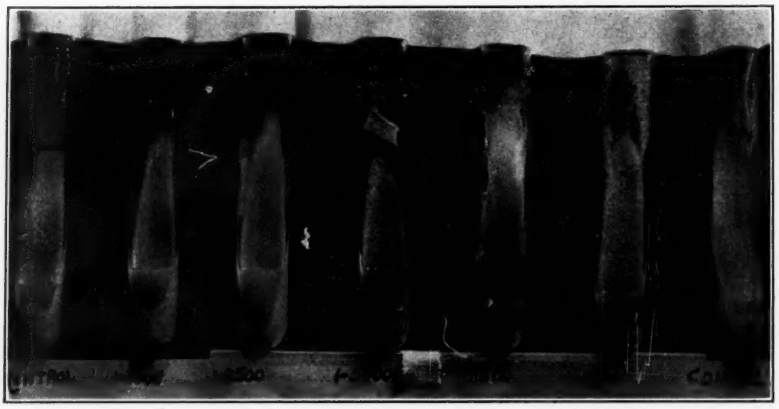

Fig. 4.—In this example from the third series of experiments two drops of light suspension of tubercle bacilli were seeded. No growth was observed in the tubes with sulfanilamide in concentrations of 1:1000, 1:2500, and 1:5000. Definite inhibition is to be noted with sulfanilamide 1:7500 and slight inhibition with 1:10,000, after three weeks.

centrations: 1:1000, 1:2500, 1:5000, 1:7500 and 1:10,000. All the control tubes showed good growths of tubercle bacilli. At three weeks no growth was observed in any of the tubes which contained sulfanilamide in concentrations of 1:1000, 1:2500, 1:5000. Growth was defi-

nitely inhibited by 1:7500 and to some slight extent by sulfanilamide 1:10,000 (Fig. 4).*

Bactericidal tests were also carried out. A suspension of tubercle bacilli made up in a buffer solution of hydrogen-ion concentration 7.2 and was mixed thoroughly with a pleural transudate of the same hydrogen-ion concentration. A smear of the transudate-bacillus mixture revealed one bacillus per 100 power oil immersion field. Clumps were not numerous and consisted of a few microorganisms. Sulfanilamide in solution was employed in the following concentrations: 1:1000, 1:2500, 1:5000, 1:7500 and 1:10,000. Samples were withdrawn at intervals up to 144 hours after incubation of the bacillus-transudate-drug mixtures at 37° C. Tubes of sulfanilamide-free media were then inoculated. At this time the sulfanilamide was estimated by the method of Marshall and was found present in the original quantities used. No bactericidal action was observed in any of the concentrations of the "drug" after incubation of the bacilli-transudate-drug mixture up to 144 hours at 37° C.

Our findings indicate that under the conditions specified sulfanilamide in certain concentrations exerts an inhibitory effect upon the growth of the tubercle bacillus in vitro. This inhibitory effect was also observed when massive doses of tubercle bacilli were employed. In line with the above mentioned facts it is worthy of note that Rich and Follis found that under the conditions of the experiments which they described, "Sulfanilamide has exerted a striking inhibitory effect upon the development of tuberculosis in the highly susceptible guinea pig." Further experiments, including animal experiments along similar lines, will be reported at a future date.

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*At six weeks a slight growth was noted in some tubes containing the drug in concentrations of 1:2500 and 1:5000.

The sulfanilamide (prontylin) employed in these experiments was generously supplied by the Winthrop Chemical Company of Canada.

THE EFFECT OF SULFANILAMIDE UPON THE DEVELOPMENT OF EXPERIMENTAL TUBERCULOSIS IN THE GUINEA PIG*

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IN A PREVIOUS communication we reported that sulfanilamide in certain concentrations exerts an inhibitory effect upon the growth of the tubercle bacillus in vitro. This inhibitory effect was also observed, but to a lesser degree, when massive doses of tubercle bacilli were employed. Bactericidal experiments gave negative results.

Rich and Follis† have stated that sulfanilamide exerts a striking inhibitory effect upon the development of tuberculosis in the guinea pig. The animal experiments which we wish to report were carried out along similar lines. It should be noted however that Rich and Follis started treatment with the drug three days before the bacilli were injected, and on the day of infection. We did not begin treatment until five and in some instances ten days following infection of the animal.

Rich and Follis reported that although a daily dose of 200 mg. of sulfanilamide would exert some inhibitory effect, more favorable results could be obtained when a total of 500 mg. of the drug was given in four divided doses, in twenty-four hours. These investigators also pointed out, however, that this dose might prove excessive, since nine out of fifteen animals in one experiment died as a result of such treatment.

In our own work eighteen guinea pigs weighing on an average 350 gm. each were infected subcutaneously in the groin with 1 c.c. of a suspension of tubercle bacilli. This suspension made up in a buffer solution of hydrogen-ion concentration 8 showed one bacillus per 100 power oil immersion field. The tubercle bacilli employed were from a four-week-old culture which was obtained directly from sputum. Five days after infection, six guinea pigs received 150 mg. of sulfanilamide twice daily. Two animals died six days after infection, that is, one day after treatment had been started; one died on the ninth day, one on the fourteenth day, and one on the fifteenth day. The remaining animal was sacrificed after forty-two days.

In a second group of six animals treatment was started ten days after infection. Two guinea pigs died early in the course of treatment, and one on the eighteenth day. The remaining animals were sacrificed six weeks after treatment had been started. The results of this experiment were suggestive but inconclusive. The local lesions were less necrotic in the treated animals. The regionary nodes too were in general smaller in the treated animals. Both treated and untreated animals showed the lungs and liver to be affected to a varying degree. We are of the

*From the Department of Surgery, Jewish General Hospital, Montreal. This work was aided by a grant from Sally Ryan.
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†Bull. Johns Hopkins Hospital 62: 77, 1938.

opinion that had the administration of the drug been by means of capsules instead of by the dropper method, and had sulfanilamide been given in smaller but more frequent doses, more animals would have survived the experiment. We regard the method of administration of the drug as an important consideration, exceeded in importance only by adequate dosage of the drug which is tolerated best if given in divided relatively small doses.

In our second series of experiments forty-one guinea pigs with an average weight of 350 gm. were infected subcutaneously, in the left side of the groin, with 1 c.c. of a suspension which showed on microscopic smear one bacillus per 100 power oil immersion field. The inoculation dose, therefore, was about fifty times less than that employed by Rich and Follis. The microorganisms used were grown for five weeks, on Petri dishes, in single colony fashion (Petroff). The tubercle bacilli employed represented the first transplant of a culture isolated directly from sputum. The animals were divided into three groups: Series I, fourteen guinea pigs, treated five days after infection; Series II, twelve guinea pigs, treated ten days after infection; Series III, fifteen untreated guinea pigs which were kept as controls. The sulfanilamide was administered orally in gelatine capsules of varied sizes (Parke, Davis & Company, No. 3, 4, and 5), at 7 A.M., 12 P.M., 6 P.M., and 11 P.M. To facilitate swallowing, the capsules were made wet in tepid water, and with the aid of an applicator were introduced in the mouths of the animals.

Series I.—Treatment of fourteen animals commenced five days after infection.

These fourteen guinea pigs were given 300 mg. of sulfanilamide in four doses of 75 mg. each on the first day of treatment to get them accustomed to the drug. On the second day, ten animals were given 530 mg. of the drug (115—150—115—150 mg.), two received 640 mg. (150—150—150—190 mg.), and the remaining two, 680 mg. (150—190—150—190). The four last mentioned animals all died within forty-eight hours as a direct result of an overdose of sulfanilamide. After only three days of daily treatment with 530 mg. of the drug, several guinea pigs appeared quite ill. It was therefore decided to suspend treatment for two days. It was then deemed advisable to reduce the total twenty-four-hour dose to 460 mg. for five animals, and to 420 mg. for the remaining five animals in this series. After treatment had been resumed for only one day, four animals died, again, we believe, as a result of too much sulfanilamide. A second two-day rest period was allowed the remaining six animals. The dose of the drug was again reduced for all animals, this time to 380 mg. Five days later, nine days after the start of treatment (fourteen days after infection), another guinea pig died. The remaining five guinea pigs then received 340 mg. (75—75—75—115 mg.) of sulfanilamide daily, for the balance of the period of the experiment. One guinea pig died after thirteen days of treatment, and two after twenty-three days of treatment. The remaining two guinea pigs were sacrificed five weeks after infection.

Comment.—No gross tuberculous lesions were distinguishable in the treated animals which died within fourteen days after infection (nine animals). In the remaining five animals which survived for three weeks or were sacrificed five weeks after infection, it was possible to observe that sulfanilamide had exerted a definite inhibitory effect upon the development of tuberculosis. This inhibitory effect was to be noted not only in the local lesions, regionary glands, and spleen, but in the other organs as well. In none of the adequately treated animals referred to was there any macroscopic evidence of tuberculosis in either the liver or lungs. The local lesions in the treated animals could not be detected in some instances. They may have been

adherent to the regionary nodes or so small as to be undiscernible. In all instances they were much less necrotic than those of the control animals. The regionary nodes were small as compared with the large indurated nodes which developed in the controls. Four of the five control animals showed multiple gross tuberculous lesions in the liver, and in three animals small lesions were noted in the lungs. The spleens of all the treated guinea pigs appeared normal in size and free from macroscopic tuberculous lesions. The spleens of the untreated control guinea pigs were much enlarged and showed multiple large areas of caseation. The spleens of the treated animals were a very dark red, while those of the infected untreated animals were light red in color.

The doses of sulfanilamide employed at the start of the above experiments may be considered excessive for our animals. Rich and Follis used 500 mg. of the drug daily for guinea pigs weighing 400 gm. We began with 530 mg. for 350 gm. guinea pigs. This dose was soon reduced to a daily dose of 460 mg., then to 420 mg., to 380 mg., and finally to a daily level of 340 mg. which was maintained. We lost eight animals early in this experiment because we gave excessive doses of sulfanilamide at the beginning. The treated animals which survived for adequate periods or were sacrificed tolerated daily doses of 340 to 380 mg. of sulfanilamide well. These latter doses of the drug are not very toxic, yet are adequate for 350 gm. guinea pigs under the conditions described. Even with such doses it is possible to interrupt treatment at intervals and at the same time obtain an inhibitory effect.

Series II.—Treatment started ten days after infection, for twelve animals.

As in Series I, these animals were given only 300 mg. (75 mg. four times) on the first day. On the second day five animals were given 460 mg. and seven animals, 420 mg. On the third day the dose was reduced to 420 mg. for all animals. On the fifth day of treatment one animal died. All animals were then given a two-day rest period. The dose of sulfanilamide was then reduced to 380 mg. On the ninth day after treatment it was decided to reduce the treatment dose to 340 mg. Eleven of the treated animals lived long enough to permit comparisons with the untreated controls; four lived to the conclusion of the experiment after five weeks.

Comment.—No gross tuberculous lesions were observed in the one animal which died fifteen days after infection. The condition of the local lesions, regionary nodes and spleens of the remaining eleven animals is appreciated best by consulting typical examples noted in the figures. (See Figs. 1, 2, and 3.) The livers and lungs of all treated animals were found free of gross tuberculous lesions. Six of ten untreated animals showed multiple macroscopic lesions in the liver, while in three, small lesions in the lungs were noted. Local lesions in the treated animals as in Series I were much smaller and less necrotic

than in the untreated controls. The regionary nodes of the treated animals were small as compared with the large nodes observed in the untreated control animals. In only one instance was the spleen of a treated animal as large as the smallest spleen of an untreated control. Otherwise, the spleens of treated animals were either normal in size or but slightly enlarged, whereas in all the untreated controls the

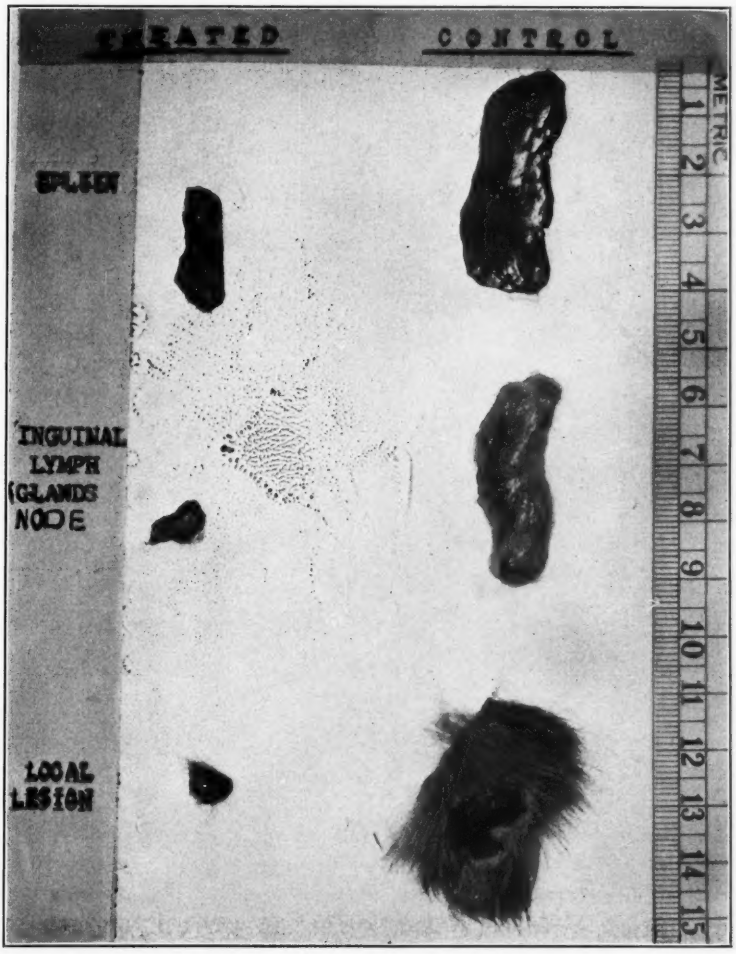

Fig. 1.—In this instance treatment with sulfanilamide was begun ten days after infection. The treated guinea pig weighed 350 gm. and received 460 mg. of sulfanilamide daily at the start. This dose proved toxic and was therefore eventually reduced to 340 mg. Treatment was interrupted at intervals. The above figure represents the findings twenty-eight days after infection, that is, eighteen days after the start of treatment. Note the large spleen, the large regionary lymph node and the more extensive necrotic local lesion of the untreated control animal. The absence of striking positive findings of tuberculosis in the case of the treated animal is not to be regarded as of exceptional occurrence under the conditions of experimentation described in the text.

spleens were greatly enlarged. Areas of caseation in the spleen were numerous and extensive in the untreated animals, small and only rarely present in treated animals. The darker spleens of the treated animals were again a striking feature.

SUMMARY

The animal experiments which are reported here indicate that under the conditions described sulfanilamide exerts an inhibitory effect upon

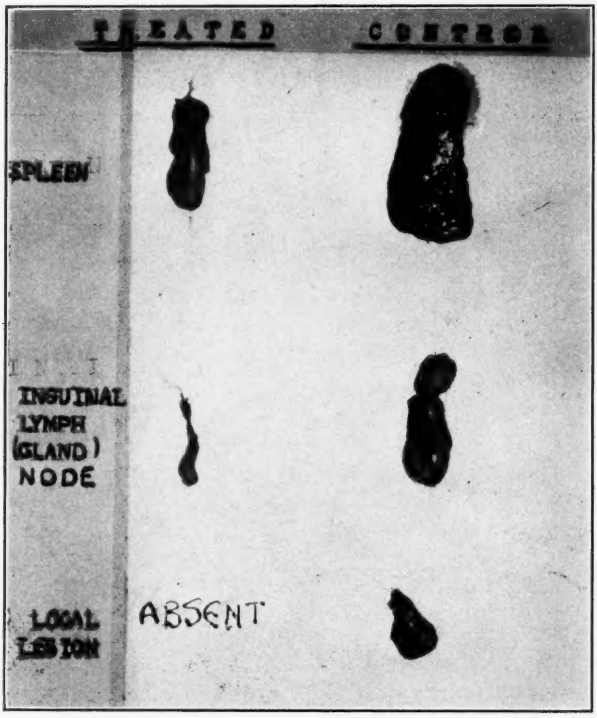

Fig. 2.—In this experiment treatment with sulfanilamide was begun ten days after infection. This guinea pig died twenty-six days after infection. Again note the small spleen and the very small regionary lymph node of the treated animal. The local lesion is recorded as absent because it could not be demonstrated grossly.

the development of tuberculosis in the guinea pig. Our findings thus confirm those reported by Rich and Follis. They may also be regarded as supplementary to and confirmatory of our own previously reported in vitro experiments.

Treatment with sulfanilamide was started five and ten days after infection. It was found that repeated relatively small doses of the drug were tolerated better than larger doses given less frequently. Daily doses of 340 to 380 mg. of sulfanilamide proved adequate yet not too toxic for most guinea pigs weighing 350 gm.

Fig. 3.—Treatment with sulfanilamide was started five and ten days after the guinea pigs were infected with tubercle bacilli. The last column of organs in the treated group is from the animal which first received treatment after ten days of infection. In this instance treatment was carried on for eighteen days; in the case of the first two it was given for twenty-three days. Note the small spleens, small regionary lymph nodes, and less extensive local lesions of the treated guinea pigs. All guinea pigs which were sacrificed or which survived two weeks of treatment with sulfanilamide showed an absence of positive findings in the organs which are illustrated. The tuberculous lesions of treated animals when present were minimal. In the case of all untreated controls tuberculous lesions were extensive and generalized.

An inhibitory effect is possible even though treatment is interrupted at intervals. This inhibitory effect is manifested as follows: the local lesion in the treated animal is less necrotic, similarly the regionary lymph nodes are less swollen and less indurated, the spleen is considerably smaller and usually free of macroscopic tuberculous lesions. In addition there is a relative absence of generalized tuberculosis in the treated animal. The untreated animal on the other hand has not only generalized tuberculosis, but in addition, the local lesion is more extensive, more necrotic, and the regionary lymph node is much larger, more swollen, and indurated. The spleen of the untreated guinea pig is not only large but also exhibits macroscopic tuberculous lesions.

Our experimental findings do not permit of clinical application.

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The sulfanilamide (prontylin) used in these experiments was generously supplied by the Winthrop Chemical Company of Canada.

TREATMENT OF ACUTE EMPYEMA THORACIS BY OPEN INTERCOSTAL DRAINAGE

A Report of 53 Consecutive Cases With No Mortality

Joseph Weinberg, M.D.
Omaha, Neb.

SIMPLICITY of operative technique in empyema becomes a consideration of utmost importance when we consider the frequency with which it is a problem for the general surgeon. Anyone who reviews the various methods of treatment which have been devised in recent years, particularly the closed methods, must be impressed by the complicated apparatus and details of management entailed in their use. Since the general adoption of the principle of a safe waiting period before operation, established by Graham and Bell,[1] it is questionable whether these intricate procedures have been more successful than the simple methods of open drainage, either with or without segmental rib resection. The fact that these complicated techniques have been advocated, however, indicates that the open drainage methods are not entirely satisfactory. During the past fourteen years I have evolved a technique, suggested by the method of Moschowitz,[2] which, in my opinion, overcomes the objectionable features of open drainage and at the same time simplifies the procedure so that it is universally applicable. This technique, a type of intercostal open drainage, has been used in 53 consecutive cases, with no deaths, and with a postoperative healing period which is shorter than that of most reported groups consisting of an equal or greater number of cases.*

METHOD

The operation consists essentially of the complete excision of the intercostal and overlying muscles between two ribs. This produces an opening which is larger than that usually obtained with rib resection. The drainage opening thus established is blocked by a tampon formed by rubber tissue packed with gauze, which permits escape of pleural exudate while diminishing ingress of air. The tampon is removed early, and the cavity is then irrigated with Dakin's solution (0.45 per cent sodium hypochlorite solution), usually without the use of tubes or other drainage material. The method does not require special apparatus.

*This report represents an uninterrupted series including all of the author's cases of empyema.

From the Department of Surgery, University of Nebraska College of Medicine.

Read at the Twenty-first Annual Meeting of the American Association for Thoracic Surgery at Atlanta, Ga., April 4, 5, and 6, 1938.

PREOPERATIVE MANAGEMENT

The time for operation is determined by aspiration of exudate from the pleural cavity at suitable intervals, usually of from several days to a week, until the density of the exudate indicates that the mediastinum and lung are fixed. In the case of most pneumococcus infections the pus is found to be sufficiently thick at the time of the first aspiration. In the case of streptococcus infections, the waiting period has been as long as three weeks. During this period the patient is maintained on a high caloric diet to allow for the increased protein metabolism known to occur with empyema (R. D. Bell[3]). Also, other general measures, including blood transfusion, are used if they are indicated.

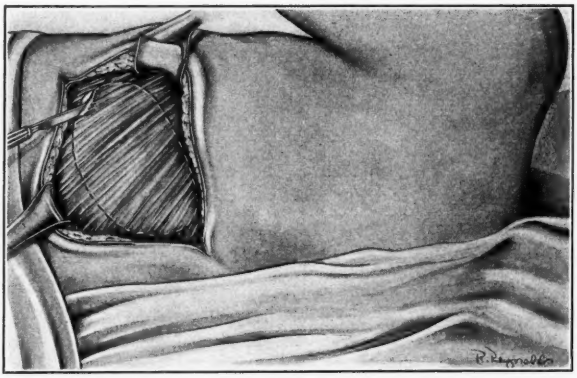

Fig. 1.—Wide excision of muscle overlying the ribs to expose the external intercostal muscle and the adjacent ribs.

OPERATION

The patient is placed on the operating table in a position which allows a maximum excursion of the uninvolved side and at the same time access to the most favorable site for drainage. Most adult patients have been operated on under local infiltration analgesia and children, under ether inhalation anesthesia. Recently, cyclopropane anesthesia has been used routinely because of the advantage of its high oxygen mixture in the presence of low vital capacity.

The site of the incision is determined by the location of the empyema, the eighth interspace posterolaterally being chosen whenever possible because of the favorable drainage from this site with the patient in the supine position, on the diseased side, and in the sitting posture. In every instance pus is aspirated at the operating table before the incision is made, even though aspiration has been performed from the same site within a few days before operation.

The cutaneous incision, usually 7 to 10 cm. in length, depending upon the size of the individual, is made along the interspace through which the pus has been aspirated. The arm should not be elevated high above the shoulder when the incision is made, otherwise the skin incision will not lie over the intercostal incision when the arm is lowered. To avoid this possibility, one may mark the line of

incision with methylene blue, with the arm lowered before making the cutaneous incision. The muscle tissue overlying the ribs is excised widely to expose the external intercostal muscle and the adjacent ribs (Fig. 1). The intercostal muscles are then completely resected between the two ribs for a distance slightly less than the length of the cutaneous incision. This is done by sharp dissection, care being taken to avoid the intercostal vessels and nerves. A small incision is then made in the

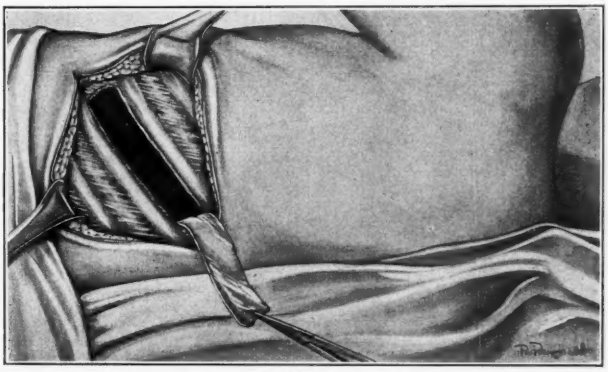

Fig. 2.—Complete excision of segment of intercostal muscles and pleura to provide adequate opening for drainage. Consult text for technique of their removal.

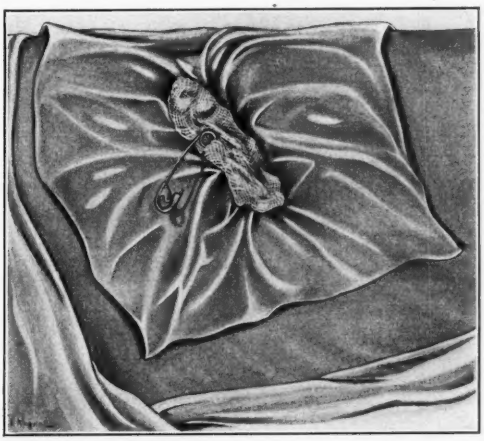

Fig. 3.—Rubber dam invaginated into the wound and held in place by large gauze packing. This minimizes the ingress of air and at the same time permits escape of pleural exudate with exhalation.

parietal pleura, and sufficient exudate is withdrawn by suction to minimize soiling after the opening is enlarged. The pleura between the ribs is then completely excised (Fig. 2). When excision is completed there should be no overhanging edges of pleura or muscle to interfere with adequate drainage. Satisfactory drainage during the period of convalescence is dependent upon the thoroughness of this excision.

The cavity is now inspected, and free fibrin masses which may be present are removed with forceps. No skin closure is made. A large piece of rubber dam, about 40 cm. square is now placed over the area with the wound in its center and is invaginated into the wound and held tightly in place by packing the pocket with a large gauze plug (Figs. 3 and 4). This effectively minimizes the ingress of air and at the same time allows escape of pleural exudate with each exhalation. It is questionable whether this safeguard is entirely necessary if there has been a safe waiting period before operation. It is believed, however, that the greatest danger of too early opening is immediately after operation and that the thoracic structures adjust themselves to moderate pressure from without after one or two days. Probably a more definite advantage of the use of rubber tissue is the freedom from pain in subsequent dressings, a consideration which is especially important when dealing with children.

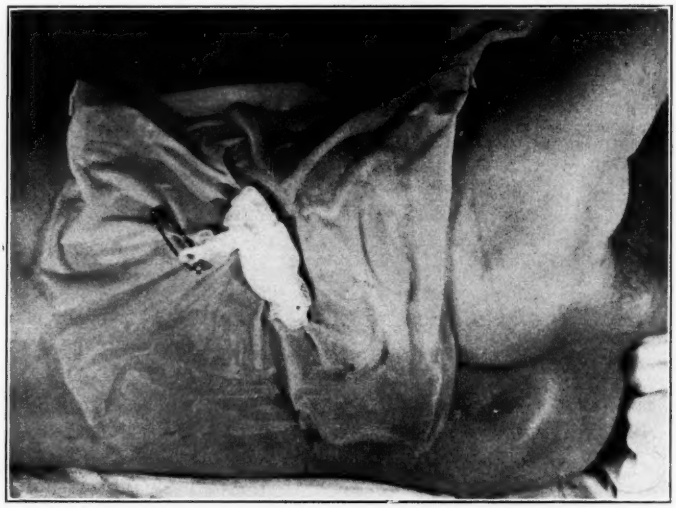

Fig. 4.—Photograph of rubber dam held in place with gauze packing.

POSTOPERATIVE MANAGEMENT

The rubber dam with its gauze plug is removed within forty-eight hours in most cases. If there is apparent respiratory embarrassment at this time, it is replaced. Replacement has been considered advisable in only a few instances. The wound is now left wide open except for a large gauze pad covering, and the pleural cavity is irrigated daily or twice daily with Dakin's solution (Fig. 5). The size of the cavity is measured at intervals of a few days to determine the progress of closure. If a bronchopleural fistula is present, an attempt is made to place the patient in such a position that irrigations can be performed without bronchial irritation. If this cannot be done, the irrigations are discontinued until the fistula is closed.

In recent cases no tubes or other drainage materials have been used. It has been found that if a sufficiently large opening is made with ad-

herence to the detail of complete removal of tags of intercostal and overlying tissue, the opening will be adequate throughout the course of treatment (Fig. 6). It is conceivable, however, that an opening necessarily made proximal to the eighth interspace posteriorly, or an opening smaller than usual because of limitations imposed by surrounding

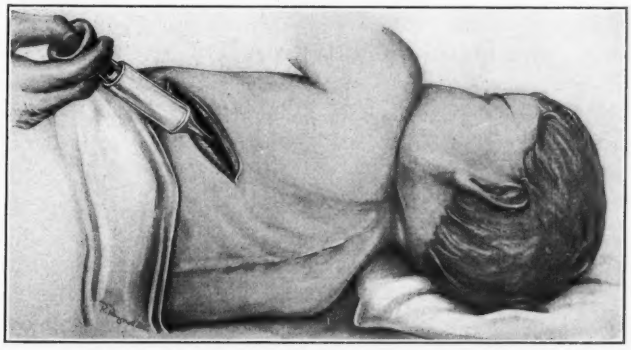

Fig. 5.—Irrigation of empyema cavity with Dakin's solution.

Fig. 6.—Photograph showing the opening six days after operation. No tubes or other drainage material are necessary to maintain adequate drainage.

adherent lung, as may occur with interlobar empyema, may require the use of long Carrel or Penrose tubes to maintain satisfactory drainage. Small pieces of gauze are never used in the dressings because of the danger of losing them in the cavity. While the use of short rubber tubes is avoided for the same reason, even more important objections

are that they may cause necrosis of the ribs with which they are in contact, or may cause damage by impinging against the lung.

The patient is encouraged to be out of bed as soon as his general condition permits, as it seems probable that activity accelerates the expansion of the lung. After a trial with various blowing devices I am not convinced that they hasten the closure of the cavity. The patient is kept in the hospital until the cavity has disappeared and all but the cutaneous wound is closed. This rule was departed from on only one occasion, and, in that instance, one in which the empyema was associated with abortion, severe anemia, and nephritis with edema, the empyema became chronic and necessitated a return of the patient to the hospital two years later for its eradication.

COMMENT

The method has been used routinely in all types of empyemas, with the exception of an anterior paramediastinal empyema which required resection of costal cartilage. The group includes a bilateral case in a child, aged 3 years, in which closure of both cavities was complete in thirty-three days, two cases of recognized interlobar abscesses in children, and one case due to ruptured pulmonary abscess.

The average healing time for all cases, with the exception of the chronic case previously mentioned, is 36.9 days. This average is based upon the number of days from operation to complete closure of the cavity. A striking difference is noted in the healing time in children and in adults. If the patients are divided into those less than 16 years old and those more than 16 years of age, it is found that the average healing time in children is 26.1 days and the average time of healing in adults is 49.6 days.

The average time of healing for the streptococcus cases is four days longer than for the pneumococcus cases. This is possibly explained by the longer waiting period before operation in the streptococcus cases. The waiting period in these cases has been perhaps longer than necessary, but it has been considered safer to risk a longer healing period than to risk a chance of mortality.

In one instance of prolonged healing the empyema was due to a ruptured pulmonary abscess. In another, delayed healing resulted from the accidental lodgment of a short Carrel tube in the cavity. After this occurrence the use of short tubes was discontinued in all cases. In three of the adults in whom there was delay in closure, phrenic nerve crush was resorted to, and this procedure greatly accelerated the closure. These patients were 48, 61, and 63 years of age, respectively.

Secondary rib resection was performed within two weeks of the primary operation in six of the early cases because of inadequate drainage

due to incomplete removal of intercostal and overlying tissue, or improper site of drainage. Since the improvement of the technique these difficulties have not occurred.

While it is acknowledged that yearly variations in virulence are an important influence in the determination of mortality and morbidity, it is also very probable that method and technique have much to do with the occurrence of complications during the period of management of the empyema. It is possible that irritation of the lung by stiff tubes, with either closed or open methods, may be conducive to the spread of septic emboli by aggravation of an already diseased lung. It is also probable that inadequate drainage after opening of the pleural cavity is a serious factor in causing complications.

TABLE I

AGE INCIDENCE

LESS THAN 2 YR.	2 TO 3	3 TO 5	5 TO 7	7 TO 10	10 TO 20
1	7	4	11	2	7
20 TO 30	**30 TO 40**	**40 TO 50**	**50 TO 60**	**60 TO 70**	**70 TO 80**
5	7	5	2	2	1

TABLE II

HEALING PERIOD—DATE OF OPERATION TO COMPLETE CLOSURE OF CAVITY

	NUMBER OF CASES	AVERAGE HEALING TIME	CHRONICITY
Under 16 years of age	28	26.1 days	0
Over 16 years of age	25	49.6 days	1
All cases	53	36.9 days	1

TABLE III

YEAR OF OCCURRENCE

Years	1924	1925	1926	1927	1928	1929	1930
No. of cases	2	1	7	3	0	1	0
Years	**1931**	**1932**	**1933**	**1934**	**1935**	**1936**	**1937**
No. of cases	4	4	6	3	9	6	7

It is believed that this method possesses the advantages of simplicity and efficiency without departing from the basic principles of treatment of acute thoracic empyema. Even the most enthusiastic advocates of the closed method must acknowledge that it is too specialized for the average surgeon who treats an occasional case and does not have the special apparatus or trained assistants demanded for its efficient administration. Other objections to the closed method are the difficulty of maintaining a closed system, the restriction of activity of the patient, the frequent plugging of the drainage tube with fibrinous masses, and the danger of dislodgment of the tube with resultant compression pneu-

mothorax. The chief objections to the open method as it is generally practiced are inadequate drainage, injury to the rib, and the danger of lodgment of foreign bodies within the pleural cavity. These disadvantages are minimized in the procedure as it is described here. I do not regard the danger of compression of intrathoracic structures from too early operation as a valid objection to open drainage, as the principles concerned with this risk must necessarily be recognized by any surgeon who undertakes the treatment of empyema. The emphasis which has been placed on simplicity in describing this method should not be misinterpreted as granting the license of carelessness in its execution. On the contrary, the method requires painstaking attention to details: a safe waiting period before operation; making a sufficiently large opening; excising all intercostal and overlying muscle completely at the site of drainage; and using care in the prevention of the entrance of foreign bodies into the pleural cavity. These requirements do not call for unusual skill but do require meticulous attention to details.

It is realized that 53 cases are not a sufficient number upon which to base an opinion on mortality rate and that there must occur instances in which associated lesions in other organs and tissues will cause fatalities. It is significant, however, that these patients, varying greatly in age, types of infection, and physical conditions, have been treated with no mortality and with a convalescent period which compares very favorably with other reported series. It has often been stated that there is no one method which is applicable to all types of acute empyemas; that some are best treated by the open method, others by the closed method, and still others by aspiration or other less frequently used procedures. Excepting the case of paramediastinal empyema mentioned, I have found this method of open intercostal drainage applicable in all cases requiring operation.*

SUMMARY

The method herein described consists of the complete excision of the intercostal and overlying muscles between two ribs for a length of 5 to 8 cm. The drainage opening so established is blocked by a tampon formed by rubber tissue packed with gauze, which permits free escape of pleural exudate while blocking ingress of air. The tampon is removed within forty-eight hours, and the cavity is then irrigated with Dakin's solution, usually without the use of tubes or other drainage material. The method is simple in its application and requires no special apparatus.

Some 53 cases have been treated in this way, cases of greatly variant ages, types of infection, and physical conditions. There has been no

*There have been several cases in which aspiration, used as a preliminary procedure, made open operation unnecessary. These cases are not included in the statistical report.

mortality, and the postoperative morbidity of consequence is limited to one case which became chronic and required a plastic operation two years later for its eradication.

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EXPERIMENTAL PULMONARY EMBOLISM

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SUDDEN death or death within a few hours, resulting from pulmonary embolism, is almost always associated with obstruction of the main pulmonary artery or its two major branches. The course of the changes in the circulation accompanying such embolism has in the past been the subject of considerable controversy. The views held have been derived in part from clinical observations and in part from experimental analysis. They can be divided into four categories, the phenomena being attributed to (1) asphyxiation,[1, 2] (2) acute right heart failure,[1, 3, 4] (3) reflex cardiac standstill,[5, 6] reflex inhibition of the respiratory center[4] or reflex vasomotor shock,[7] (4) obstruction to the flow of blood to the left heart with a consequent reduction in its output.[1, 8, 9] Gibbon and others[8] attribute this reduction in output to a pooling of blood in the systemic veins behind the obstruction with a consequent reduced volume of circulating blood.

Pulmonary vascular obstruction has been produced experimentally (1) by occluding the main pulmonary artery or its two major branches by ligation or external compression[1, 8, 10-12] and (2) by injecting particulate objects intravenously.[2, 4, 13, 14] While the first of these methods imitates clinical massive pulmonary embolism, it has the disadvantage of requiring an extensive surgical approach. The second method entails the obstruction of many smaller branches, which Binger and others[15] have shown is associated with respiratory and circulatory changes other than those occurring with occlusion of the main pulmonary artery. The chief difference is that in the latter there is a reduction of flow to all parts of both lungs rather than a cessation of flow in isolated portions.

Blood clots were used experimentally in the past, but in many instances they were broken up in the heart and produced disseminated small emboli in the lungs. Mann[16] had to introduce as many as eight to twelve clots before his animals died. Martin[17] attempted to form a clot in situ in the femoral vein making it radiopaque by barium sulfate impregnation, but succeeded in getting a riding embolus of the major branches only once. Hall and Ettinger[9] were more successful in producing riding clots and embolism of the main pulmonary

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artery. They prepared their clots in vitro with formalin and introduced them into the jugular vein. In many of their experiments, however, many of the smaller peripheral pulmonary branches were completely occluded by small clots. Neither Martin nor Hall and Ettinger made detailed studies of the circulatory changes.

It was felt, therefore, that a detailed study of the circulatory changes following experimental embolism of the main pulmonary artery or both its branches was essential to evaluate the probable mechanism operating in this type of clinical pulmonary embolism. Our major problem was to develop a method of producing such endovascular pulmonary obstruction. A priori considerations and preliminary trials soon convinced us that the embolus had to be (1) wide enough to occlude the major pulmonary vessels, (2) light and pliable enough to be easily propelled by the blood stream through the veins and past the tricuspid and pulmonary valves, and (3) long enough so that the tail of the embolus could partially obstruct the main pulmonary artery or pass into the other main branch to form a riding embolus. In man, the femoral vein, especially if varicose, is large enough to form a thrombus sufficiently long, thick, and pliable to obstruct the main pulmonary vessels. In the dog, neither the femoral nor external jugular veins are wide enough to permit the production of sufficiently thick thrombi or the introduction of such thrombi or other objects to obstruct the major pulmonary vessels consistently. After considerable experience, however, a method was devised by which endovascular obstruction of the main pulmonary artery or its major branches could be produced in 92 per cent of trials.

PROCEDURE

The embolus consisted of Penrose tubing filled with fluid. It was prepared as follows: A piece of tubing 13 cm. long and 9 mm. in diameter was ligated twice at one end with silk ligatures and the end trimmed and inverted over a long hemostat so as to have the exterior surface smooth. The other end of the tubing was tied onto one of the horizontal limbs of a glass T-tube. A long flexible, lead-tipped, rustless steel wire was inserted into the Penrose tube through a rubber tube attached to the other horizontal limb of the T-tube in order to keep the Penrose tubing taut, and the wire was held in place by a hemostat clamped on the rubber tube. A 20 c.c. Luer syringe containing 15 c.c. of a dilute barium sulfate mixture was attached to the perpendicular limb of the T-tube, and the mixture was injected into the Penrose tube to test for leaks. All the air and fluid contained in the Penrose tube was then drawn back into the syringe, and the barium sulfate mixture free of air was reinjected in a quantity sufficient to give the required turgor, the quantity injected being noted. It was usually about 5 c.c. Before inserting the Penrose tubing in-

travenously, a few c.c. were withdrawn and a second hemostat clamped on the rubber tubing connecting the syringe with the T-tube. The arrangement at this point is shown in Fig. 1.

Following this the right jugular vein of a dog weighing from 16 to 29 kg., anesthetized with nembutal, and lying on its back, was exposed in the neck, and the cephalic end was tied off. The vein was opened between this point and a tautly drawn ligature below and held open by two mosquito clamps. The Penrose tube moistened with

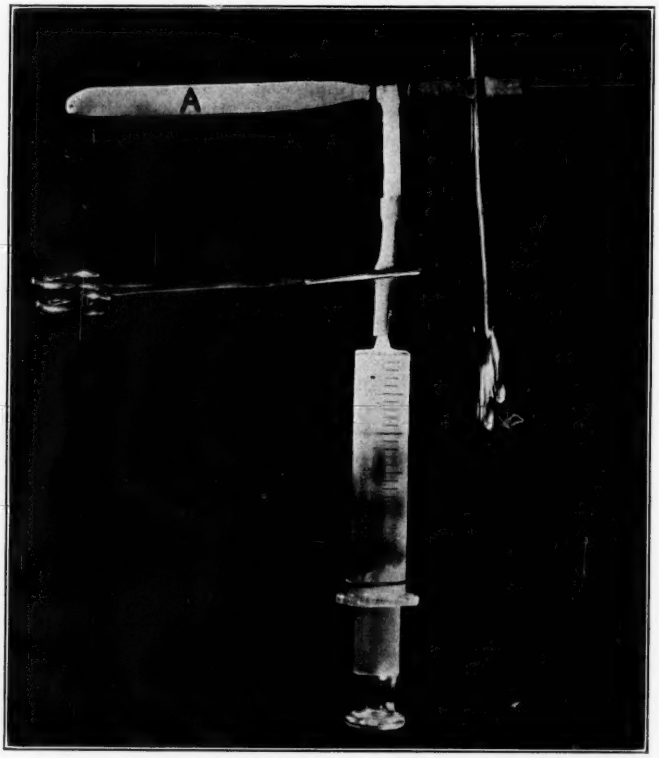

Fig. 1.—Photograph of the arrangement before insertion of the embolus into the vein. *A* is the Penrose tubing embolus.

saline solution was inserted through this opening and past the distal ligature which was carefully loosened. Sometimes, in order to facilitate the insertion, it was found necessary to loosen the right paw, depress the vein, or lift the head, while manipulating the tube gently with the wire. The tube was always inserted up to its attachment to the T-tube, at which time the steel wire stilet was carefully withdrawn and the hemostat reapplied. The fluid in the Penrose tube was then completely withdrawn into the syringe, and after loosening the liga-

ture, the previously determined quantity for proper turgor was reinjected. The tail of the Penrose tube embolus was next tied off with two silk ligatures and cut loose above the ties. The nose of the embolus now projected into the superior vena cava and was able to take up the additional distention caused by the constricting effect of the relatively narrow jugular vein. The embolus was then pushed into the vein with a large hemostat and the vein temporarily closed above the tube by traction on the loose ligature. A glass cannula was now tied into the vein through which 150 c.c. of isotonic saline solution was forcibly injected in order to propel the embolus into the circulating blood stream. The vein was then tied off. We learned that this propulsion was facilitated when the animal was turned on the left side. The position of the embolus was easily checked by fluoroscopy and the fluoroscopic findings confirmed in every case by autopsy.

The circulatory changes were determined in each animal by comparing a series of observations before with a similar series after this procedure. The observations included the standard limb lead electrocardiogram, pulse rate, systemic mean arterial and venous blood pressures, cardiac output, blood volume, hemoglobin content, ether and cyanide circulation time, rectal temperature, and total plasma solids. The technique for carrying out these procedures is described in a previous communication,[18] except for the total solids which were determined gravimetrically.[19]

A control series of animals were similarly analyzed in order to evaluate the effect of the entire procedure including anesthesia. In these control animals all the steps in the procedure were carried out as described above and in the same time sequence, except that the embolus was withdrawn after its insertion into the vein and before the injection of 150 c.c. of saline solution. Autopsies were done on these control animals, particularly to rule out the presence of hemopericardium, which sometimes followed right heart puncture.

RESULTS

Massive pulmonary embolism was successfully produced in fifteen experiments.* Nine of these emboli were found riding both pulmonary branches. In three experiments the embolus was in the main pulmonary artery and one of its major branches. (In one of these there was a fibrin plug in the right pulmonary branch, which was probably a true embolus derived from the jugular vein or the auricle and may have prevented the artificial embolus from subsequently entering this branch.) In two experiments the embolus obstructed the main pulmonary artery and pulmonary conus, and in the remain-

*In three of these the embolus had been introduced into the right femoral vein instead of the right jugular. The femoral vein was soon abandoned because the embolus frequently was caught at the auriculocaval angle as a result of the change in the direction of the blood stream at this point. The route via the superior vena cava is less angulated.

ing experiment it almost completely occluded the right and partially obstructed the left and main pulmonary arteries. The circulatory observations were successfully completed in eight of these experiments, in four of which there were riding emboli.

After the first successful embolism, there were nine failures out of twenty-four trials, excluding, of course, experiments which were discarded due to extraneous technical accidents, such as hemopericardium, overanesthesia, etc. In these nine failures the emboli were caught in the superior or inferior vena cava or in the right auricle. When the technique was perfected, there was only one such failure in twelve trials, a success of 92 per cent.

Seven of the fifteen animals with massive pulmonary embolism died spontaneously at intervals varying from fifteen minutes to three hours after the introduction of the embolus. In four of these death was unquestionably due to the embolus alone; in the other three the possibility of contributory causes, such as excessively deep anesthesia and cyanide, could not be excluded. It is noteworthy that none of the five control animals died in this period. The remaining eight animals were sacrificed at intervals ranging from one to three and one-half hours after introducing the embolus. No doubt some of these would have eventually died spontaneously, had they not been sacrificed.

The usual course of events after the introduction of the embolus was as follows: For a period varying from three to thirty minutes the pulse was grossly irregular, due, at least in those experiments in which electrocardiograms were taken, to auricular and ventricular premature systoles from multiple foci. At the same time, the respirations often became more rapid and sometimes deeper. The irregular pulse persisted indefinitely in those experiments in which the embolus remained lodged in the auricle and was found there post mortem. In the others, apparently at the time when the embolus passed into the pulmonary artery, the irregular pulse was superseded by a regular, more rapid and weakened pulse. At the same time, the respirations usually became more rapid and more shallow. Cyanosis was difficult to estimate but was unquestionably present in several experiments, particularly terminally. Gasping respirations were the sign of impending death, and in one experiment there was profuse pulmonary edema.

Fluoroscopy was valuable in determining the success of the experiment. Typically, the embolus was seen projecting beyond the heart shadows on both sides, especially in the oblique positions. Fig. 2 shows a typical riding embolus.

At necropsy the embolus was found covered with a fibrin coating. In addition, marked edema of the lungs with frothy fluid in the air

Fig. 2.—Roentgenogram of a riding embolus in the pulmonary arteries. No barium is seen at the junction where the Penrose tube is folded.

Fig. 3.—Photograph of specimen showing a riding embolus in the pulmonary arteries. The arrow indicates the embolus from which the fibrin has been washed for better contrast.

passages was found in one experiment, and moderate congestion with occasional hemorrhages was found in several others. In the rest, no abnormalities were seen grossly. No histologic examinations were made. Fig. 3 is a photograph of a typical riding embolus.

The data of the circulatory observations in the embolism experiments are assembled in Table I and those for the controls in Table II. In addition, the changes in cardiac output are plotted in Fig. 4. The changes in the control experiments recorded in Table II fall within the range for controls found in our previous work on a larger series.[18]

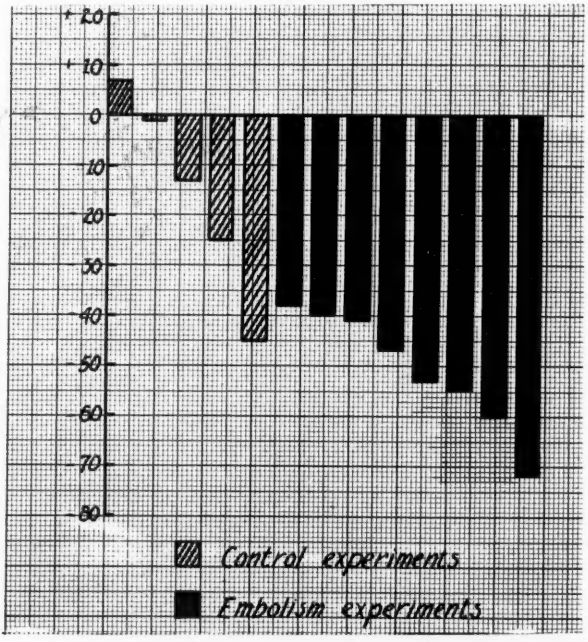

Fig. 4.—Changes in cardiac output, after the procedure, in embolism experiments as compared with control experiments. The average percentage decreases in cardiac output and blood volume were: embolism experiments, 50.4 per cent, cardiac output, and 0.7 per cent, blood volume; control experiments, 15.1 per cent, cardiac output, and 3.2 per cent blood volume.

Following pulmonary embolism the minute volume output of the heart was found to be definitely decreased, in nearly all instances, beyond that in the control series (Fig. 4); and the average decrease in output was definitely greater than the average in the controls. The decrease in stroke volume, because of the development of tachycardia, was even more marked. This change in cardiac output was accompanied by no consistent change in oxygen consumption but was always associated with an increase in the arteriovenous oxygen difference.

In several instances an arterial anoxemia developed. On the average, the mean arterial blood pressure fell more and the venous pressure rose slightly more in the embolism than in the control experiments. No consistent changes in ether circulation time were observed, although in two instances it was increased beyond the experimental error. There

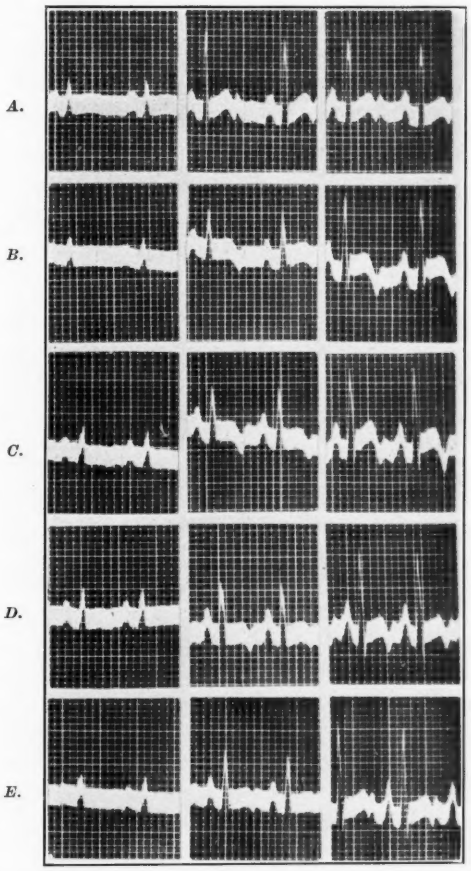

Fig. 5.—Three lead electrocardiograms to show changes which accompany pulmonary embolism. *A* was taken before and *B*, *C*, *D*, and *E* at twenty-minute intervals after embolism. For discussion see text.

was usually a moderate increase in the cyanide circulation time. No significant change in rectal temperature occurred.

The circulating blood volume was not significantly altered by the embolism, the changes in most instances being slight and of the same order as in the control experiments. In several of the embolus experiments, however, there was an increase in total circulating blood volume

TABLE

EMBOLISM

EXPERIMENT	18 ♂		21 ♀		27 ♂	
Weight (kg.)	24.5		27.3		29.3	
Time of observation in relation to introduction of embolus	before	1 hour after	before	1 hour after	before	1½ hours after
Pulse rate (beats per minute)	180	225	165	195	150	180
Oxygen consumption (c.c. per minute)	156	143	164	169	153	158
Arterial blood oxygen (volume per cent)	17.89	20.51	16.23	18.73	16.61	14.26
Venous blood oxygen (volume per cent)	13.70	14.33	12.91	12.94	13.03	7.31
A-V oxygen difference (volume per cent)	4.19	6.18	3.32	5.79	3.58	6.95
Minute volume (c.c. per minute)	3723	2312	4909	2919	4245	2273
Stroke volume (c.c.)	20.8	10.6	29.8	14.9	21.2	12.6
Cell volume (c.c.)	918	940	1414	1332	986	845
Plasma volume (c.c.)	900	883	1305	1225	1420	1300
Total blood volume (c.c.)	1818	1823	2719	2557	2406	2145
Hemoglobin per cent	110	111	89	103	78	80
Mean arterial blood pressure (mm. Hg)	148	122	140	142	138	140
Venous blood pressure (cm. water)	7	3	8	30?	–12	–2
Ether circulation time (seconds)	4.4	3.1	4.1	5.2	4.2	5.2
Cyanide circulation time (seconds)	10.2	18.1	9.0	13.0	9.2	13.2
Total plasma solids (gm. per 100 c.c.)	8.59	8.01	7.20	6.56	8.50	8.00
Rectal temperature (°C.)	39.5	40.0	39.2	39.7	38.7	38.0
Remarks	Sacrificed in 2½ hours. Riding embolus. Small patch of pneumonia, lower left lobe.		Died in 3 hours. Riding embolus.		Sacrificed in 3½ hours. Embolus obstructing left branch completely, right branch and main pulmonary artery partially.	

associated with an increase in cell volume. This is attributed to a compensatory constriction of the spleen with a discharge of its erythrocyte-rich contents, the result of the anoxia following the decreased blood flow and the arterial anoxemia.[20] The hemoglobin concentration changes, as expected, followed qualitatively those in the ratio of cell to total blood volume. There were no significant variations in plasma solids, indicating the absence of any significant loss of plasma fluid.

In all but two experiments there were electrocardiographic changes of a character not encountered in the controls. In most experiments there was an elevation of the S-T segment in Leads II and III, most marked in Lead III, with an inversion of the T-waves in these leads (Fig. 5). In one experiment, there was a depression of the S-T segment in Leads II and III, the T-waves, which were inverted before embolism, becoming upright. These changes superficially resemble those seen in experimental coronary occlusion, although it is to be noted that the S-T segment in Lead I changes in one experiment in

I

EXPERIMENTS

31 ♂		41 ♂			42 ♂		50 ♂		51 ♂	
23.2		18.4			18.2		17.3		16.1	
before	1 hour after	before	¾ hour after	1¾ hours after	before	1½ hours after	before	1¾ hours after	before	2½ hours after
184	198	144	172	184	124	168	136	176	172	144
136	123	123	159	137	111	139	108	114	141	152
18.72	18.13	12.71	19.50	10.47	13.50	17.60	14.84	17.53	11.45	12.74
15.92	11.75	10.57	14.92	6.48	12.12	11.50	11.29	9.68	9.54	8.12
2.80	6.38	2.14	4.58	3.99	1.38	6.10	3.55	7.85	1.91	4.62
4857	1930	5747	3469	3434	8050	2279	3040	1453	7375	3293
26.4	9.8	39.8	20.2	18.8	65.0	13.5	22.4	8.3	42.9	22.9
1256	1370	684	1066	1065	849	870	597	711	677	585
1165	1140	1270	1200	1045	1505	1305	1000	1025	1325	1200
2421	2510	1954	2266	2110	2354	2175	1597	1736	2002	1785
88	92	69	81	100	62	80	73	80	62	65
134	174	170	120	140	130	100	150	130	120	110
9	13	–1	4	2	–1	4	10	–8	–2	–10
4.3	11.3	4.4	3.8	4.2	7.5	7.5	?	7.2	3.0	6.0
13.2	45.3	9.4	12.8	13.0	?	11.6	6.8	12.2	7.5	10.0
6.21	6.01	7.19	6.66	6.98	7.78	7.14			7.34	7.25
38.0	37.5	39.0	39.0	39.0	38.5	38.5	38.0		39.0	39.2
Died in 1¼ hours. Embolus folded double, obstructing main pulmonary artery and conus.		Died in 2 hours with pulmonary edema. Embolus obstructing right pulmonary branch and partially left.			Sacrificed in 2 hours. Riding embolus.		Sacrificed in 2½ hours. Embolus obstructing left pulmonary branch, main pulmonary artery, and conus.		Sacrificed in 3 hours. Riding embolus.	

the same rather than in opposite direction to that in Lead III. Left axis shift, characterized by the appearance of an S-wave in Leads II and III making the QRS diphasic, was found in several experiments. There was also some decrease in QRS amplitude in several experiments (Fig. 5). It is noteworthy that in several instances the S-T and T-wave changes were transitory even when the circulatory disturbances were progressive (Fig. 5).

DISCUSSION

The picture of experimental massive embolism of the major pulmonary arteries can be depicted from our experiments, provided that it is recognized that positive findings in some experiments are more significant than their absence in others. This is so because we are dealing with a condition varying in intensity and in the rate of its progress in the different experiments. There is also a varying balance between the degree of obstruction, with its train of consequences on the one hand and the compensatory mechanisms set up on the other. If the

TABLE II

CONTROL EXPERIMENTS

EXPERIMENT	32 ♀		34 ♂		36 ♀		38 ♂		39 ♀	
Weight (kg.)	19.1		19.1		20.5		20.9		23.9	
Time of observation in relation to manipulation of embolus	before	1½ hours after	before	1 hour after	before	2 hours after	before	1½ hours after	before	1 hour after
Pulse rate (beats per minute)	176	168	144	160	228	200	132	168	160	180
Oxygen consumption (c.c. per minute)	112	115	94	97	147	192	101	146	111	152
Arterial blood oxygen (volume per cent)	14.57	15.00	17.34	19.00	19.92	21.78	18.30	19.35	15.54	18.20
Venous blood oxygen (volume per cent)	13.06	12.21	13.49	14.99	15.93	16.91	13.78	11.81	12.47	12.60
A-V oxygen difference (volume per cent)	1.51	2.79	3.85	4.01	3.99	4.87	4.52	7.54	3.07	5.60
Minute volume (c.c. per minute)	7417	4122	2441	2418	3684	3942	2216	1936	3610	2713
Stroke volume (c.c.)	41.1	24.7	16.9	15.1	16.2	19.7	16.9	11.5	22.6	15.1
Cell volume (c.c.)	937	889	841	1010	666	729	1155	1110	954	863
Plasma volume (c.c.)	1355	1325	1215	1175	935	910	1225	1110	1325	1125
Total blood volume (c.c.)	2292	2214	2056	2185	1601	1639	2380	2220	2279	1988
Hemoglobin per cent	78	81	85	90	96	102	98	103	86	90
Mean arterial blood pressure (mm. Hg)	130	160	130	140	144	154	105	155	175	150
Venous blood pressure (cm. water)	+8	–2	–7	–7	+12	+12	–2	–14	–2	+3
Ether circulation time (seconds)			7.2	8.4	3.2	3.3	6.5	4.5		5.6
Cyanide circulation time (seconds)	6.2	7.1	15.2	14.2	6.2	8.4	14.4	14.6	12.8	11.2
Total plasma solids (gm. per 100 c.c.)	6.68	6.56	6.96	6.80	6.51	6.47	7.13	7.10	7.91	7.23
Rectal temperature (°C.)	38.5	37.5	38.5	37.5	39.0	39.0	38.5	38.0	37.5	38.0
Remarks	Sacrificed in 2½ hours. No gross abnormalities.		Sacrificed in 1¾ hours. No gross abnormalities.		Sacrificed in 3 hours. No gross abnormalities.		Sacrificed in 2½ hours. No gross abnormalities		Sacrificed in 2 hours. No gross abnormalities	

compensatory mechanisms are adequate, most of the handicaps of the obstruction are soon overcome, and little or no demonstrable circulatory disturbances will ensue. Furthermore, it must be realized that it was not possible to obtain more than one or, at most, two sets of complete data after the obstruction, and in some instances some of the changes which later led to the animal's death might have developed after the last set of detailed observations were made.

Bearing these considerations in mind, on the basis of these experiments we can present the following summary of the effect of massive pulmonary embolism in the first few hours following the introduction of the embolus. The extremes, of course, are no demonstrable changes when the obstruction is too small and death in fifteen minutes in the most severe case. As the embolus passes through the heart it causes the appearance of extrasystoles from multiple foci, presumably by mechanical irritation. The entrance and lodgement of the embolus in the pulmonary artery is heralded by the appearance of a regular tachycardia and an apparent diminution in the pulse amplitude. The respirations become accelerated and more shallow. There is a reduction in cardiac output per minute and a greater reduction in stroke volume with little or no change in the oxygen consumption and an increase in the arteriovenous oxygen difference. Cyanosis and arterial anoxemia may develop later. The change in arterial and venous blood pressure is variable, although there is usually a moderate fall in the former and a slight rise in the latter. The change in ether circulation time is also variable although it is occasionally definitely increased, and the cyanide circulation time is usually moderately increased. There is no change or a slight increase in blood volume (due to an increase in cell volume) and no perceptible loss of plasma fluid. Death, preceded by agonal respirations, may occur at intervals varying from fifteen minutes to three hours after the introduction of the embolus. At autopsy congestion, and occasionally pulmonary edema, can frequently be demonstrated.

This picture is complete enough to evaluate the pre-existing theories of the mechanism of changes set up by pulmonary emboli. Our data do not support the neurogenic theories and are in accord with the evidence on this point presented by Hall and Ettinger.[9] Since death was not instantaneous but took place at least several minutes after embolism, it is unlikely that we are dealing with reflex cardiac standstill or reflex inhibition of the respiratory center. There is also no evidence of vasomotor collapse, since, if anything, a reflex vasoconstriction must occur as a part of the compensatory mechanism tending to prevent the arterial blood pressure from falling. We have no evidence bearing on the suggestion of Peiser[21] that reflex spasm of the pulmonary artery augments the obstruction caused by the embolus.

In the absence of denervation experiments, the evidence on all these points must continue to remain indirect.

As regards the other three concepts, namely, obstruction, asphyxia, and heart failure, they are in reality facets of the entire complex associated with massive pulmonary embolism. The primary factor is in the last analysis the obstruction itself which, because of the increase in resistance it interposes, dissipates some of the energy of the right heart and thereby leads to a reduction in the output of this chamber and hence also that of the left heart. In the absence of any change in total circulating blood volume, this reduction in cardiac output must be associated with a redistribution of the blood in the circulatory bed. The absence of a fall in arterial pressure commensurate with the decrease in cardiac output indicates a decrease in the capacity of the arterial bed resulting from vasoconstriction of the peripheral arteries. The only remaining regions where the circulation can be slowed and the blood can accumulate, therefore, are the capillaries and the veins. The fact that the circulation time from vein to carotid sinus (cyanide) is not retarded to the same extent as the minute volume flow indicates indirectly in the presence of an unchanged circulating blood volume that the retardation of blood flow and accumulation of blood take place chiefly in that part of the circuit not included in the circulation time measurements, namely, the systemic capillaries and peripheral veins. That the lung capillaries probably also share in this accumulation of blood is indicated by the fact that the cyanide circulation time is usually increased in the absence of any change in the ether circulation time. Moreover, congestion and in one instance pulmonary edema were found post mortem. Such pulmonary findings could be explained by assuming failure of the left ventricle, which we have no reason to believe occurred early in the course of the experiment. The pulmonary capillary dilatation most likely occurred because of anoxia of the lung tissue. It is important to emphasize that our experiments show that no pooling of blood took place in the sense of a loss of circulating blood volume. We are not dealing, therefore, with either surgical shock or vasogenic (histamine-like) shock, since both of these conditions are characterized by such a loss of blood volume.[22, 23]

There is no evidence pointing to acute heart failure as a contributory cause early in the sequence of events. Heart failure may develop secondarily as a result probably of a deficient coronary blood flow aided in some instances by arterial anoxemia. The electrocardiographic changes support this view. That acute failure of both heart chambers can occur is evident from experiments in which the pulmonary artery is completely occluded by external compression.[24] Such heart failure, in our experiments, however, is a complication occurring late in the more complete occlusions. That some dilatation of the

right heart probably does occur is apparent from the occasional rise in venous pressure observed. In most instances, however, the right heart can cope with the increased resistance aided by the decreased inflow and in this way prevent the venous pressure from rising.

Arterial anoxemia is also a late complication and, like heart failure, contributes to the downward course of the animal. The arterial anoxemia is probably the result of the impediment to oxygen transfer from the alveoli to the blood. This is probably due to the presence of fluid in the alveoli and to changes in the alveolar wall caused by the deficient pulmonary circulation.

From the foregoing discussion it is apparent that the primary factor in all these changes is the obstruction itself which causes a reduction in minute volume flow and a redistribution of blood in the circulatory system. All the other phenomena encountered are sequelae of this, and death is eventually due to anoxia of the heart, of the respiratory center, or of both. We are, therefore, dealing with a type of hypokinetic circulatory failure (Blalock[25]) distinct from others hitherto described. It is neither neurogenic, hematogenic, vasogenic, or cardiogenic. It is due to a major obstruction in one portion of the double circuit which comprises the mammalian blood-vascular system. Hence, it is a form of shock *sui generis*.

SUMMARY

1. A method is described for consistently reproducing massive embolism of the major pulmonary arteries in the dog.

2. Such embolism, it was found, is associated with (1) a decrease in cardiac output and arterial blood pressure, (2) an increase in cyanide circulation time, (3) an increase in the heart and respiratory rates, (4) sometimes with an arterial anoxemia, probably due to impairment of the permeability of the pulmonary membranes, (5) frequently with an unchanged or an increased circulating blood volume, attributable to a discharge of erythrocytes from the spleen, (6) usually with permanent or transient changes in the S-T segment and T-waves of the electrocardiogram and (7) usually without significant changes in venous pressure, ether circulation time, oxygen consumption, temperature, or total plasma solids.

3. Death in this type of pulmonary embolism, when it occurs, is probably caused by anoxia of the brain or of the heart with secondary acute heart failure, or both. This anoxia is attributable primarily to the decrease in cardiac output and secondarily in some experiments to arterial anoxemia.

4. The decrease in cardiac output in massive embolism of the large pulmonary arteries is caused by a major obstruction in one portion of

the double circuit which constitutes the mammalian blood-vascular system, and therefore represents a hitherto unemphasized distinct form of shock.

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TUBERCULOSIS OF THE INTERCOSTAL LYMPH GLANDS: LYMPHATIC DRAINAGE OF THE PLEURA

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THE abundant lymphatic supply of the pleura is well known. Most of the studies of the lymphatics of the pleura have shown their presence in the visceral pleura and the diaphragm with their subsequent drainage into the hilum nodes. There exist, however, much fewer data as to the channels through which drainage from the parietal pleura may pursue its course to gain its ultimate goal, the thoracic duct, or the right lymphatic duct. Kampmeier believes there is the possibility of devious anastomotic lymph channels, created particularly where adhesions of the pleurae exist. The recent work of Simer and Webb has shown the rapidity with which these new channels develop. In their studies of the adhesions between the jejunum and the omentum experimentally produced in dogs, they demonstrated newly formed lymph channels which were traced through a series of cut sections. As early as eight days after operation India ink injected into the jejunum near the site of the adhesion appeared in these new formed vessels.

Interest in these lymphatics was first aroused by finding enlarged intercostal lymph nodes during the course of a thoracoplasty for pulmonary tuberculosis. It had been the habit of Hedblom to include in this procedure a crushing of the intercostal nerves. In the isolation of these nerves frequently an enlarged node was encountered and removed for study. No attempt was made to search for these glands, and those which have been studied were found incidentally during the aforementioned procedure or during the ordinary process of stripping the periosteum from the rib prior to its resection.

The parietal lymph glands consist of three groups: the sternal, the intercostal, and the diaphragmatic. The sternal group lies along the internal mammary artery and receives lymph from the adjacent chest wall as well as from the deeper structures of the anterior abdominal wall, above the level of the umbilicus.

The intercostal or vertebral glands lie along the intercostal vessels and occupy these spaces near the vertebral column. They drain the posterolateral region of the chest, and the upper spaces lead to the right lymphatic duct on the right side while the left joins the thoracic duct. The diaphragmatic glands lie on the thoracic aspect of the

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diaphragm and are grouped in three sets, anterior, middle, and posterior.

There are two sets of lymphatic vessels of the thoracic wall, a superficial one associated with the musculature and mammary glands, and a deep one which includes the drainage of the intercostal muscles and the pleura.

Miller confirmed the contention of Sappey that the pleural lymphatics are arranged in an abundant meshwork of irregular polyhedral rings. The existence of stomata in the pleura was an early conception of von Recklinghausen, but this view is no longer accepted.

Grossly, the removed glands have varied in size from 0.4 to 2.3 cm. in their longest axis. The average size has been $1.2 \times 0.8 \times 0.6$ cm. in its three dimensions. The color may be red brown, pink gray, or black, or some combination of these. Most of them were found along the processes of the spine and the angle of the rib. They, however, were also seen in the midaxillary line.

The microscopic sections of the majority of the glands have been carefully studied by Edwin F. Hirsch, whose pathologic description is used here. The glands were sectioned at several levels, in some instances as many as twenty-three sections being made of one node. This proved of value in that some areas of the same gland showed normal lymphatic tissue, hyaline scars, or chronic inflammatory reaction while others showed the typical histologic structure of a tubercle.

One gland consisted of a mass of lymph tissue with carbon pigmentation and several hyaline scars. This patient had an adherent upper lobe and a four-year history of tuberculosis.

Another node had the usual structure except that in addition there were focal masses of chronic granulation tissue, histologically tubercles. They were oval or circular structures with a fibroblastic stroma supporting epithelioid cells, some lymphocytes, and occasional Langhans' giant cells.

Another node was pigmented with carbon and had focally a few oval and round structures, histologically tubercles, that consisted of newly grown connective tissue, epithelioid cells and a few multinucleated giant cells. The centers of some of these were necrotic, which represented caseation.

There was a total of 45 glands removed from 32 patients. The average duration of the disease was slightly more than three and one-half years. The presence of adhesions was determined through evidence as shown by the x-ray plate or the failure to find a free space on attempted pneumothorax. In nine cases a partial pneumothorax still existed, and in one of these serous fluid was present, and five were complicated by empyema. Two of these were sterile, and the other three were secondarily infected and had coexisting drainage of the pleural cavity at the time of the thoracoplasty.

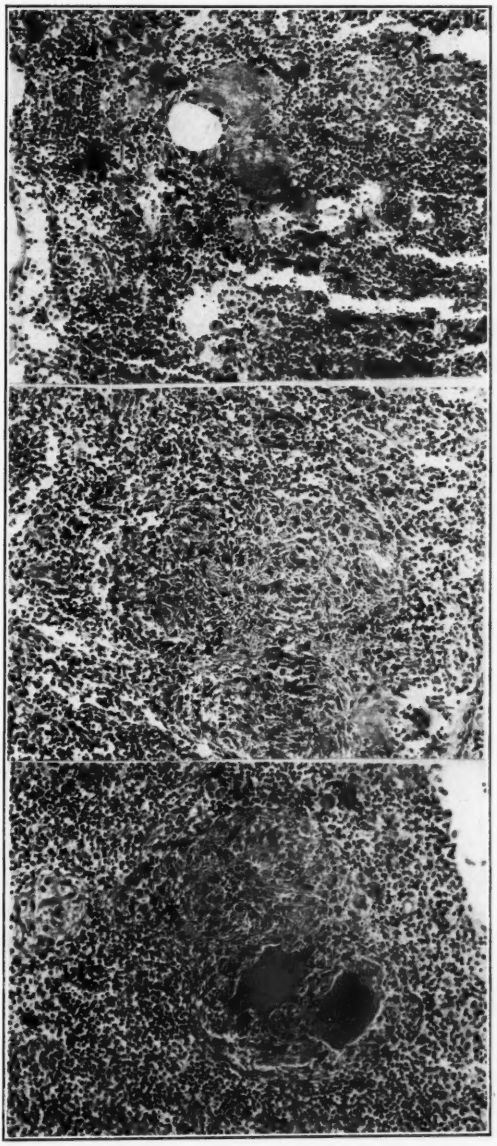

Fig. 1.—*A*, Section of normal lymphoid structure with hyaline scars and abundant carbon pigmentation.

B, Section of another node showing focal masses of chronic granulation tissue, histologically tubercles. The fibroblastic stroma supporting epithelioid cells, lymphocytes and occasional Langhans' giant cell are seen.

C, Section of a third node shows some carbon pigmentation with focal areas of new grown connective tissue, epithelioid cells, and multinucleated giant cells.

In seventeen cases the adhesions were considered extensive, and partial in twelve, while in three their presence or extent was not known. Twenty-eight glands showed typical histologic evidence of tuberculosis in the sections examined; ten were normal lymphoid tissue, and seven showed inflammatory changes which could not be said to be tuberculosis.

TABLE I

NAME	DURATION YEARS	ADHESIONS		PNEUMOTHORAX PRESENT	SEROUS FLUID	PYOPNEUMOTHORX		GLAND—MICROSCOPIC			
		EXTENSIVE	PARTIAL			STERILE	SECONDARY INFECTION	NORMAL STRUCTURE	CARBON PIGMENTATION	CHRONIC INFLAMMATORY CHANGE	HISTOLOGIC TUBERCLES
L.F.	2	?		No							++
E.M.	6	+		Yes			+	+	+		
O.C.	5		+	No				+	++		++
R.B.	7	+		No							++
D.P.	4	+		No					+	+	
A.H.	1½	+		No				+			+
M.C.	2		+	No						+	
E.W.	4½		+	No				+	+	+	
J.L.	5	+		Yes				+	++		
R.O.	2½	+		Yes					+		
A.M.	3	+		Yes			+				+
W.G.	2		+	No				+			
M.B.	1½	+		Yes			+		+		++
N.V.	4	+		Yes	+						+
C.B.	2½	+		No							+
M.S.	3		+	Yes		+				++	++
H.E.	12	+		No				+	+		+
G.T.	3	+		No				+	+		+
A.C.	1½		+	Yes						++	
B.L.	3	?		No							
A.G.	2		+	No							+
D.G.	3		+	Yes		+		+	+		
L.L.	2½		+	No					+		++
P.W.	3		+	No					++		+++
H.M.	7	+		No					+		+
G.M.	3	+		No							+
L.C.	4	?		No				+	+		
B.B.	7	+		No					+		+
V.H.	2½		+	No							+
A.A.	1		+	No							+
D.A.	2	+		No							+
H.H.	10	+		No							

In eighteen of the glands carbon pigmentation was observed in the examined sections, and this occurred in the normal gland as well as in those showing inflammatory reaction.

The direct absorption from the pleural cavity by the parietal pleura is rather obvious and could account for the changes observed in the intercostal glands. It is interesting that two of the normal glands occurred in empyema cases. One was secondarily infected while the other was sterile, and in the aspirated pus of the latter many tubercle bacilli were demonstrated.

Because some nodes revealed chronic inflammatory reaction on their removal, they were bisected at the operating room table; one portion was used for microscopic examination, and the other portion was macerated in a mortar under aseptic conditions and cultured in ordinary culture media. There was no growth from any of eight glands so cultured. One deep infection occurred in a patient in whom a gland was removed, but examination of this gland showed a normal lymph gland without inflammatory changes.

Bull called attention to the origin of infection in thoracoplasty from injury to infected tissue in the periosteal stripping. Deep seated infections do occasionally occur and may give rise to dyspnea and toxemia, and days may pass with no evident signs of the true nature of the condition even after careful examination of the wound. Two such cases have been observed. Though the glands which showed they communicated with an infection in the lung parenchyma by their chronic inflammatory changes failed to show organisms when cultured, yet it is conceivable that injury to the underlying tissue could open up these lymph channels and thus give rise to such a complication. Churchill reported the removal of a mediastinal lymph gland in the course of a lobectomy, and it too failed to grow organisms on culture, though an empyema subsequently developed which may have had its origin as he suggested from the focus drained by this node. He records the experience of Auchincloss who removed mesenteric nodes with a subsequent streptococcic peritonitis. It seems probable then that trauma to the underlying tissue can be the origin of a deep seated infection.

The microscopic descriptions given are representative of the entire group. In some merely hyaline scars are observed which may indicate some absorption from the pleura or from the lung underneath and adherent visceral pleura; while in others there has been a transmission of carbon pigmentation, tuberculous infection, and changes due to nonspecific infection in the lung. These hyaline scars may result from inflammatory disease or be the reaction of the gland to carbon pigment.

A demonstration of the chronic inflammatory changes of nonspecific origin and the histologic tubercles in these nodes might be assumed to be due to an involved pleura as seen where adhesions exist, or pleural involvement, as demonstrated by fluid or empyema. The presence, however, of carbon pigmentation which must come from the lung parenchyma would indicate the establishment of definite lymphatic communications to these glands. If this assumption is true, then it is likely that the other changes previously described are likewise induced through these same newly created channels, and do not necessarily indicate specific tuberculous disease of the pleura, though of course it may exist. The absence of tuberculous pleural involvement adjacent to adhesions to be severed is frequently observed in thoracoscopic examinations.

Lemon and Higgins used finely divided graphite to demonstrate the lymphatic pathways of absorption from the peritoneal cavity through the diaphragm. Later they used the same particulate matter to show its absorption from the pleural cavity. The mediastinal and sternal nodes were found most active, while the sternal, vertebral and diaphragmatic lymph nodes showed some absorption. There was no evidence of passage of the particulate matter through the visceral pleura into the lung.

The carbon pigment as observed in the studies of these glands is comparable to the graphite used in the experiments of Lemon and Higgins. These authors were unable to demonstrate a lymphatic current coursing toward the periphery but did observe that the primary flow of the particulate matter stopped as soon as the lymph glands were choked with the graphite.

Scott and Beattie compared the distribution of tuberculous lesions of the lymphatic glands and vessels of the thorax and upper abdomen in man and other primates, and concluded that the system in all is essentially the same and the parietal glands consist of three groups; a sternal group which is the most important and a vertebral and diaphragmatic group. They also found lymphatic vessels formed in pathological adhesions in serous cavities and ventured that the tendency of adhesions to form might be attributed to infection of the subpleural or superficial lymphatic system. A reversal of flow of lymph may exist when a blocking of the tracheobronchial and bronchial pulmonary nodes occurs.

Tuttle and Womack used a carmine suspension to demonstrate the flow from the periphery to the hilum nodes by way of the deep lymphatics. They further concluded that particulate matter was carried to those nodes more rapidly from the periphery than when injected in the wall of a major bronchus in spite of the proximity of the latter to the hilum. Their studies were made in connection with bronchiogenic carcinoma, and they concluded that carcinoma peripherally situated had a more rapidly fatal course by earlier extension through these lymphatics or the blood stream. Diagnostic pneumothorax has often illustrated the presence of adhesions in neoplasms of the lung. Evidence of chest wall involvement, in glands, the invasion of the soft tissues, or adjacent bony structure, is still too commonly seen when the diagnosis of bronchiogenic carcinoma is first established. The mechanism of this involvement may be suggested by these studies.

On two occasions during the course of a phrenic operation a gland lying in the areolar tissue over the scalenus anticus muscle has been removed, and in each instance an abundance of carbon pigmentation and histologic tubercles were demonstrated in the cut sections, indicating the communicating pathways to those cervical nodes as well as the intercostal glands.

CONCLUSIONS

Microscopic sections of intercostal lymph glands may reveal histologic tubercles or nonspecific inflammatory changes and the presence of carbon pigmentation.

The presence of carbon pigmentation indicates a communication with the lung parenchyma. The transmission of this pigment is a reversal of the normal lymph flow.

There is experimental evidence of newly formed lymph channels where adhesions exist in serous cavities.

Newly created channels may account for the changes described.

Trauma to these lymph channels could conceivably give rise to a deep seated infection.

Dissemination of tuberculous infection may occur through these newly created anastomotic channels. Likewise metastatic carcinomatous lesions may spread in this manner.

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A METHOD FOR PREVENTING AND CONTROLLING SUBCUTANEOUS EMPHYSEMA FOLLOWING CLOSED INTRAPLEURAL PNEUMOLYSIS*

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SOME subcutaneous emphysema probably occurs following every closed intrapleural pneumolysis. This complication is serious in only a small percentage of cases (approximately 3 per cent). When large amounts of air escape into the thoracic wall through the trocar openings, the artificial pneumothorax may be difficult to maintain or may even be lost, especially if the pneumothorax is a small one at the time of pneumolysis. More often leakage of air from the pneumothorax and frequent fillings necessary to maintain it lead to the development of pleural fluid, occasionally followed by an obliterating pleuritis. Rarely air may become encapsulated under the skin when the trocar opening results in a permanent sinus between the subcutaneous tissues and the artificial pneumothorax. In other patients the extension of the subcutaneous emphysema along the arm, into the face, or over the abdomen has been annoying, without causing permanent or serious consequences, as long as the pneumothorax is controlled.

The purpose of this paper is to describe a method and a rationale for the control and prevention of subcutaneous emphysema, with its sequelae, following closed intrapleural pneumolysis.

Apparently air from the pleural cavity penetrates, via the trocar openings (which may remain patent for days), parietal pleura, endothoracic fascia, and intercostal muscles before spreading to the tissues, where it is clinically demonstrable. The extension of air through the tissues not only varies according to its pressure, amount, and available supply, but also is conditioned by the permeability and contiguity of the interaponeurotic surfaces. Air injected subcutaneously in dogs at 25 cm. of water pressure was readily forced into the tissue spaces lying just above the rib cage but not into intercostal or endothoracic spaces lying deeper in the chest wall.† The plugging of the trocar openings at the level of the ribs should prevent escape of air from the pleural cavity following closed intrapleural pneumolysis, provided that no other openings into the intercostal muscles or mediastinum have been made by the operative procedure.

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†Unpublished data.

The usual single skin suture does not approximate the defect in the intercostal musculature. The ordinary methods of applying an adhesive dressing over the trocar wounds do not tend to plug the trocar opening at the level of the ribs and may even raise the skin and underlying tissues away from the opening, thus allowing the air an easier avenue of escape from the pleural cavity. Therefore the adhesive strapping is placed so as to produce marked skin tension over the trocar wounds (Fig. 1).

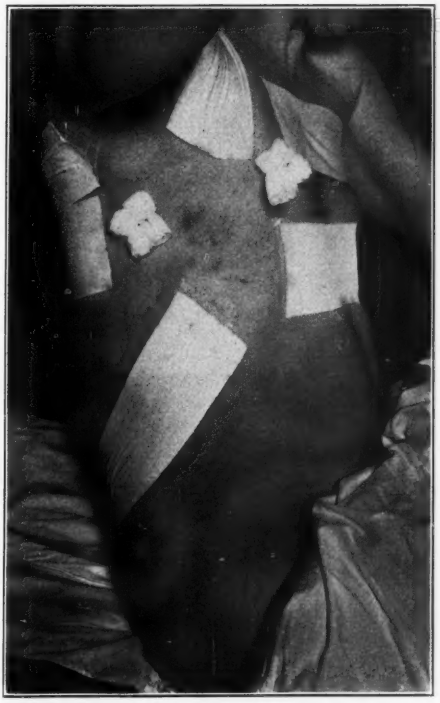

Fig. 1.—Skin tension adhesive dressing applied after closed intrapleural pneumolysis. Note that the adhesive strappings are applied so as to stretch the skin tightly against the chest wall in that area where the trocar openings were made.

The control of the intrapleural pressure is important to the prevention of subcutaneous emphysema. After an open pneumothorax is closed at the height of expiration, the resulting intrapleural pressures are more negative than after closure during inspiration.[1] In order to leave a negative pressure in the pneumothorax cavity after closed intrapleural pneumolysis, the open trocars are removed at the height of expiration.

Cough is a potent cause of subcutaneous emphysema. During the expiratory phase of cough the intrapleural pressure becomes highly positive[1] and air is forced through the trocar opening from the pleural

cavity. It becomes important, therefore, to allay cough both during and following the closed intrapleural pneumolysis.

The actual technique for preventing and controlling subcutaneous emphysema after closed intrapleural pneumolysis consists of:

1. Preoperative sedation with nembutal and morphine is administered in adequate doses.

2. The trocars are removed at the height of expiration, and the skin is lightly massaged.

3. A single skin stitch of dermal or silk tied over gauze bolsters is placed with a large curved Keith needle through each trocar opening.

4. Skin tension adhesive dressing is applied (Fig. 1).

5. An anteroposterior x-ray film of the chest is made immediately after the patient leaves the operating room.

6. If indicated from the x-ray film, fluoroscopy and artificial pneumothorax, with adjustment of intrapleural pressures, are performed immediately.

7. Codeine phosphate or morphine sulfate is given hypodermically every four hours to allay postoperative cough.

CASE 1.—B. D., a 43-year-old man, S. F. H., No. 224622, had an artificial pneumothorax March 17, 1937, and closed intrapleural pneumolysis was performed July 23, 1937. One adhesion was completely cut and another broad palisade adhesion partially cut. Ordinary dressing was used. Immediately after, subcutaneous emphysema occurred over trocar wounds. The next morning there was subcutaneous emphysema in right side of thorax, face, and in axilla. Pneumothorax fillings were required to maintain collapse. Fluid developed to dome of the diaphragm. Skin tension dressing was used and no further increase in subcutaneous emphysema noted. On August 13, 1937, a second closed intrapleural pneumolysis was done. One large palisade adhesion was cut. Skin tension dressing was applied and no palpable subcutaneous emphysema developed. Fluid did not increase.

CASE 2.—J. D., a 37-year-old male, S. F. H., No. 224271, had an artificial pneumothorax started on the right Nov. 10, 1936, and closed intrapleural pneumolysis was performed July, 1937. There was cold dissection in endothoracic fascia of one large band adhesion. Ordinary dressing was used. Immediately after, subcutaneous emphysema of face, neck, thorax, axilla, arm, and finger occurred, progressing. Skin tension dressing was used after twenty-four hours. There was no further progression. Artificial pneumothorax filling on the evening of operation and next day was necessary for maintenance of adequate pneumothorax. Fluid developed to dome of diaphragm. Thoracoscopy was performed August 6, 1937. No adhesions were cut. Skin tension dressing was used and there was no subcutaneous emphysema and no further fluid. On September 15 thoracoscopy was performed. No adhesions were cut. Skin tension dressing was used and no subcutaneous emphysema occurred.

CASE 3.—B. C., a 31-year-old woman, S. F. H., No. 219660, had a right artificial pneumothorax March 24, 1937, and right closed intrapleural pneumolysis was performed May 21, 1937. Eight string and band adhesions were cut. Ordinary dressing was used. There was moderate subcutaneous emphysema over upper right thorax

and neck. On the evening of the pneumolysis there was artificial pneumothorax, 500 c.c. After twenty-four hours there was no further extension of subcutaneous emphysema. Fluid developed to dome of diaphragm. On July 21, 1937, closed intrapleural pneumolysis was performed. One thick band adhesion was partially cut and three string adhesions were cut. Skin tension dressing was used. No palpable subcutaneous emphysema developed. Fluid disappeared in ten days. Immediate artificial pneumothorax was not necessary for adequate collapse.

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AN UNUSUAL ACCIDENT OCCURRING IN CLOSED INTRAPLEURAL PNEUMOLYSIS

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IN THE past few years the operation of closed intrapleural pneumolysis, as originated by Jacobeus, is deservedly coming into wide use. With the improvement in instruments and technique the early sad accidents of hemorrhage, rupture, empyema, etc., have become so rare that the great value of the results of a successful pneumolysis now far outweigh the dangers involved. It is now generally recognized as an indispensable addition to the armamentarium of the specialist in tuberculosis.

Welles reported on 141 cases in which closed pneumolysis was done for unsuccessful collapse of cavity (J. THORACIC SURG., August, 1932). Of these patients, 122 or 87 per cent showed satisfactory collapse of cavity following the operation; 6 or 4 per cent developed purulent effusion without mixed infection; 2 or 1.5 per cent developed mixed infection, both of these patients recovering. Welles and Woodruff, in a series of 66 cases during the year 1934, reported that 74 per cent (62) of those who had cavity before operation were apparently closed following the operation. In 4 or 7 per cent closure could not be determined. In the entire series only two deaths occurred which were in any way attributable to pneumolysis (presented to N.T.A., 1936). Wells and Woodruff report in several hundred cases since 1932, on which statistics have not been compiled, that no other accident such as I am reporting has occurred (personal communication). Peters and Cornish (A.R.T. January, 1936) reported its use in 88 of 244 unsuccessful pneumothoraxes. Cavity closure was obtained in 78 per cent.

Certainly favorable reports of this kind will result in even more general use of the procedure. The results are so definite and so immediate that there can be no dispute as to its value, and there can be no doubt that closed pneumolysis is a permanent part of collapse therapy in suitable cases.

Considering the increasing use of closed pneumolysis it seemed to the writer that the occurrence of an unusual and dramatic accident in one of his cases would be worth reporting, since no similar accident has been found in the literature.

In reviewing the history of the patient the following facts are noted:

A female patient, single, aged 24 years, presented herself to me in May, 1933. The family history was unimportant, and the past history was negative as regards tuberculosis. Early in October, 1932, she was diagnosed as tuberculous and treated

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at home until her arrival at Saranac Lake in May, 1933. On admission she had disease disseminated throughout both lungs, with a large cavity (6 cm.) in the right apex and a small cavity, (2 cm.) in the left apex. Because of extensive bilateral disease no collapse therapy seemed advisable, and she was treated with bed rest alone until the spring of 1935. At this time the left lung had become apparently healed, and pneumothorax was begun on the right, with an unsuccessful partial collapse. String and bandlike adhesions were present, preventing collapse of the large cavity. In August, 1935, the Jacobeus operation was performed, at which time several adhesions were severed, with no closure of cavity resulting. Over the following winter one remaining adhesion at the apex was stretched sufficiently by continued pneumothorax refills so that pneumolysis was again attempted on Feb. 11, 1936. A thick, short adhesion, running to the apex, was successfully severed, the operator wedging into the parietal pleura with cautery. The patient stood the operation very well. During the course of the operation a trickle of blood was observed running down the inside of the thoracic wall at the point of the entrance of the cautery cannula. There was never at any time bleeding from the adhesion. The patient helped herself from the table to the stretcher and was in good condition, when she gave a sudden sharp cough, immediately became cyanotic, and lost consciousness. Auscultation of the chest showed no evidence of shift of the mediastinum, but the heart sounds were very loud and roaring, the characteristic sound of air in the heart. This peculiar roaring sound lasted three or four minutes and gradually subsided, the heart sounds becoming perfectly normal. At the same time the patient regained consciousness and complained of severe dyspnea. For several days dyspnea and nausea were present. After the first twenty-four hours the dyspnea occurred in spells of an hour or two with comparative comfort in between. Edematous râles were present over the entire left lung for several days, with much frothy expectoration for thirty-six hours, the typical picture of acute pulmonary edema. The temperature rose to 102°F., gradually subsiding to 100°F.

On the eighth day she was discharged from the hospital and put on sanatorium care. Cough and expectoration steadily became worse, the temperature remained above 100°F., dyspnea gradually increased, and death occurred on March 12, 1936. X-ray films of the chest one week before death showed spread of disease in lower half of left (contralateral) lung. An autopsy was performed and revealed the following:

Right Lung:—There was partial collapse and a cavity 5 by 3 by 3 cm. in the apex. Adhesion stump was smooth and showed no evidence of oozing. There were isolated foci of fresh aspiration disease in the lower lobe.

Left Lung:—Fresh aspiration disease was widespread through the lower lobe; Nothing else of interest as bearing on the cause of death was found in the rest of the body.

Summarizing the facts of the accident following the second operation, the chain of events seems to me to be quite obvious. In inserting the cautery cannula, a good-sized blood vessel was punctured in the chest wall, as shown by the bleeding at this site. The slow nature of the bleeding would lead me to speculate that this was the intercostal vein which was punctured. On withdrawal of the cannula the vein was still open, and the sudden sharp cough immediately afterward forced considerable air into it from the pleural cavity. At no time was there any bleeding from the adhesion stump, so that this was the only possible point of entrance for air into the blood stream. The air entered the right heart, coming through the peripheral circulation. As this was pumped from the

heart into the lungs through the pulmonary artery, multiple air emboli occurred in the left lung, as shown by the dyspnea and subsequent pulmonary edema.

The patient died from aspiration of infectious material into the lower lobe of the collapsed right lung and the lower lobe of the left lung. This was the result of the loss of consciousness and subsequent pulmonary edema and extreme dyspnea. Whether the shock to the lung tissue of the multiple air emboli and pulmonary edema played a part in this can only be inferred. Microscopic examination of the lungs shows only clusters of small acute aspiration tubercles surrounded by zones of pneumonic reaction and some slight hemorrhage into the air spaces.

The writer has seen one other case in which pulmonary tuberculosis was present, where multiple air emboli in the lung resulted from a faulty blood transfusion apparatus. In this case at least 20 c.c. of air was introduced into the median basilic vein. While dyspnea and pulmonary edema resulted, there was no activation of the apical tuberculosis. However, the tuberculosis was not extensive and was fairly well healed. In the present case reported, the disease was widespread, and while it had shown extensive healing by x-ray examination, and had been stationary for many months, the presence of so much disease must have meant that the balance of resistance was very finely drawn, so that a localized lowered resistance of only a few days' duration may have been enough to turn the balance against the patient. This, of course, is purely theoretical.

No treatment is suggested other than supportive. Such an accident is obviously extremely rare, and, if it does occur and death does not ensue immediately from shock or suffocation, the end result on the contralateral lung depends on the amount of disease therein and its condition, or the occurrence of aspiration.

While such an accident has been known to be possible theoretically, so far as can be ascertained it has not been reported, and it should be known as one of the rarer hazards of closed pneumolysis.

DISCUSSION

DR. W. W. WOODRUFF.—The technique used in this case did not differ from the usual. The lysis went very smoothly. As a matter of fact, Dr. Leetch and I had both left the room. The patient raised herself from the operating table and helped herself over to the stretcher. The nurses were putting on her jacket. She suddenly coughed and fell back unconscious. I do not believe there is much doubt as to the diagnosis of air embolism. The only question is as to the site of entry. My guess is that it must have been through the intercostal vein rather than through the adhesion. With the cannula going through the intercostal space, the vein could be wounded and then occluded by the cannula. When the cannula was withdrawn, it seems possible the clot may have been disturbed, allowing air to enter; but since the vein was thin walled, one would not expect it to remain patent. Against any explanation, many arguments can be brought forward.

DR. SMITH.—Did you take a positive pressure?

DR. WOODRUFF.—When the wound is closed, we make no attempt to increase the pressure, and it is practically atmospheric. I very seldom have the patient take a deep inspiration before withdrawing the cannula because even if it does give them a positive pressure temporarily, the excess air soon leaks into the subcutaneous tissues.

DR. EDWARD S. WELLES, Saranac Lake, N. Y.—I think Dr. Woodruff's and Dr. Leetch's explanation of the mechanism which caused this is undoubtedly correct. I cannot figure any other way out. It is possibly similar to the case that Dr. Jacobaeus mentioned yesterday, in which he had some hemorrhage into the chest from the chest wall, and the case that I spoke of yesterday in which a rather large hemorrhage occurred, enough to almost fill the chest in the hours following the operation, which I am sure came from leakage of the intercostal vein, in the very same manner that Dr. Woodruff has just mentioned.

The vein was kept closed by the presence of the cannula. Withdrawal of the cannula removed the clot, leaving the open vein, which went on bleeding. In this, instead of the vein going on bleeding, the air from the cough apparently rushed into it. I do not see any other way to explain it, but it is certainly a very interesting observation.

DR. JULIAN A. MOORE, Asheville, N. C.—I do not recall in the review I made of pneumonolysis several years ago, a report of pulmonary embolism, but there was one reported patient with cerebral embolism in that series who died.

DR. HENRY W. LEETCH, Saranac Lake, N. Y.—Since there was no bleeding from the visceral stump, we could not see how air could have entered the pulmonary circulation. The clinical picture that we received was air embolus in the peripheral circulation. In the pulmonary circulation an air embolus would have given us a very different picture.

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